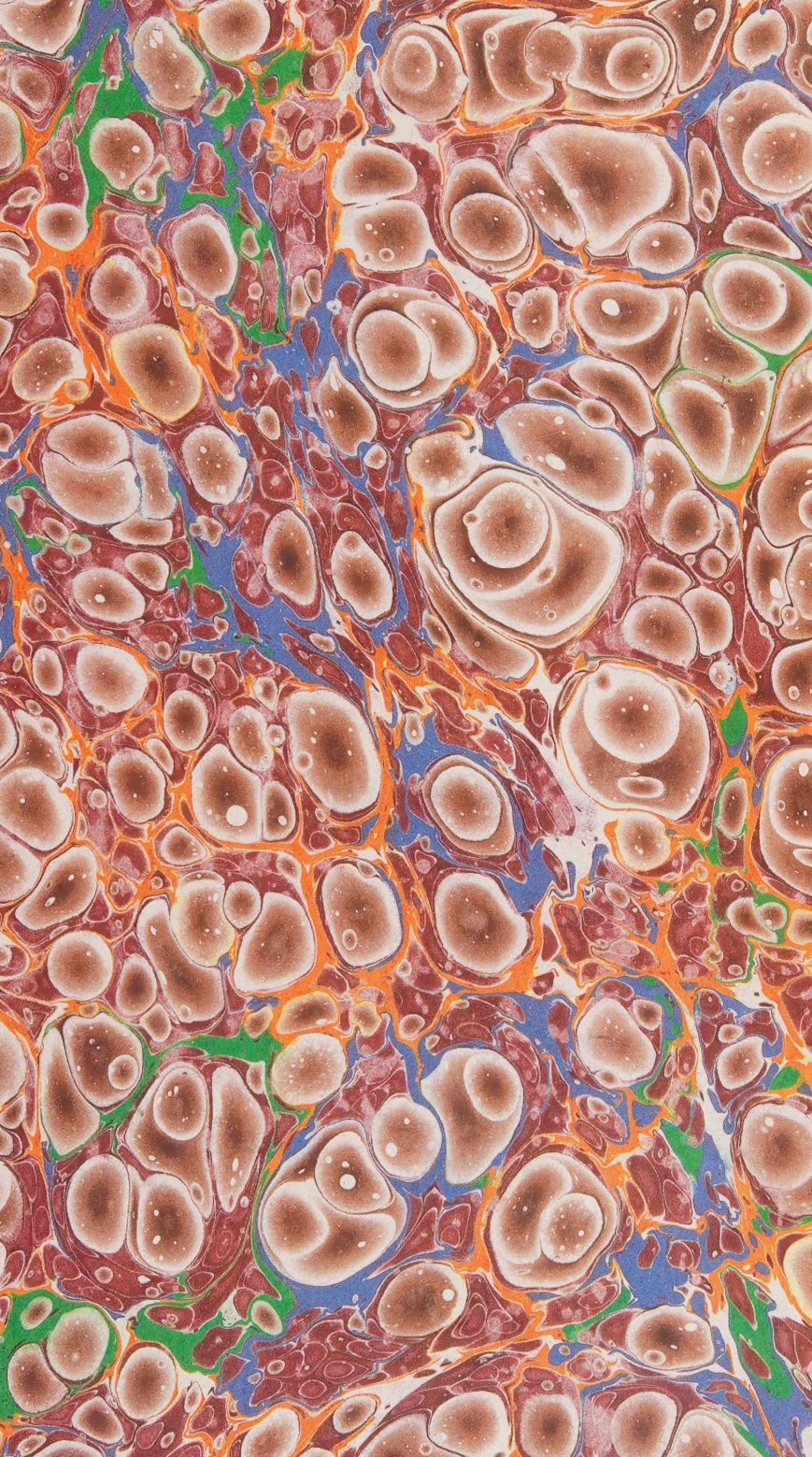






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THIS SPECIAL EDITION OF
ON THE USE OF
THE OPHTHALMOSCOPE
IN DISEASES OF
THE NERVOUS SYSTEM
AND OF
THE KIDNEYS;
ALSO IN CERTAIN OTHER GENERAL DISORDERS

By Thomas Clifford Allbutt



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THE OPHTHALMOSCOPE

CLIFFORD ALLBUTT

‘How may a man know whether he be so earnest is worth enquiry; and I think there is this one unerring mark of it, viz. the not entertaining any proposition with greater assurance than the proofs will warrant.’

LOCKE, *Human Understanding*.



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CONGESTION PAPILLA
(Enlarged 15 Diameters)

ON THE
USE OF THE OPHTHALMOSCOPE
IN DISEASES OF
THE NERVOUS SYSTEM
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BY
THOMAS CLIFFORD ALLBUTT, M.A., M.D. CANTAB.

FELLOW OF THE LINNEAN SOCIETY;
FELLOW OF THE SOCIETY OF ANTIQUARIES;
FELLOW OF THE ROYAL MEDICAL AND CHIRURGICAL SOCIETY;
PHYSICIAN TO THE LEEDS GENERAL INFIRMARY,
AND LECTURER ON PRACTICE OF MEDICINE,
ETC. ETC.

London and New York
MACMILLAN AND CO.

1871

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OXFORD:

BY T. COMBE, M.A., E. B. GARDNER, AND E. PICKARD HALL,

PRINTERS TO THE UNIVERSITY.

TO

J. HUGHLINGS JACKSON, M.D., F.R.C.P.

MY DEAR DR. JACKSON,

It was about the same time, I think, that you in London and I in Leeds began to use the Ophthalmoscope in the investigation of cerebral diseases; and it was not among the least of its many uses to me that it procured me the pleasure and the advantage of your friendship. Your simplicity, truthfulness, and acuteness in the observation of disease, and your genuine insight into facts so long obscured by the cumbrous and vain phraseology of the Schools, have made your friendship as valuable to me as a student of nature, as the like qualities in your personal character have won my warm regard as your friend.

I cannot but feel that this volume should have come from your hand and not from mine. Circumstances, however, of many kinds, your encourage-

ment being among the chief of them, have determined me to publish my own experience in this form : I have given my best pains and care to make my little book of some worth ; to you, to whom I dedicate it, to the illustrious Von Gräfe, who has passed away, and to all those whose work has enlightened this dark subject, must be given the first fruits of my reward. *Nisi enim alii ante nos impedimenta removissent, via nobis libera et expedita haud esset ; ita partem gloriolæ cujusque nostræ haud exiguum debemus aliorum, qui ante nos eadem tentaverant, laboribus.*

Believe me, my dear Dr. Jackson,

Yours very faithfully,

T. CLIFFORD ALLBUTT.

38, PARK SQUARE, LEEDS,

1871.

PREFACE.

SOME considerable parts of the present volume have already been published. I may refer more especially to my paper upon the 'Optic Nerves and Retinas of the Insane,' which appeared in the volume of 'Transactions of the Medical and Chirurgical Society' for 1868; to an article on 'Medical Ophthalmoscopy,' which appeared in the number of the 'British and Foreign Medico-Chirurgical Review' for January, 1868; to some lectures on 'Optic Neuritis,' which appeared in the 'Medical Times and Gazette' between the 9th of May and the 1st of August, 1868, inclusive; and to some papers on the use of the Ophthalmoscope in tubercular meningitis and in spinal disease, which appeared in the 'Lancet' of May 1st and May 8th, 1869, and of January 15th, 1870. Little or nothing, however, has been transferred to this book as it stood. I have found it necessary to revise the whole, and to re-write large portions, in order to add the results of more experience and to express maturer opinions. Thus a great part, the far greater part indeed of this volume, is now issued for the first time; and its pages might have been almost indefinitely multiplied had I thought it right to use all my accumulated material, and to publish a large number of cases. I have thought it better, however, to exercise much reserve in this matter, and to publish my own conclusions, with a few illustrative cases

only, rather than to print a mass of such evidence as may be collected for himself by any practitioner who is interested in the subject.

It is with some reluctance that I have forborne to publish a larger number of coloured drawings with the text. To execute drawings of any value, however, is a very laborious and a very costly task; one which I would not have avoided had it been a necessary one, but which is, I think, less necessary now that so many useful plates have been published by the leading writers on diseases of the eye, and now that Liebreich has published an English edition of his invaluable Atlas under the able management of Mr. Swanzy. Moreover, it seemed clearly undesirable to increase the cost of a volume like this, which, as it contains an account of the work of many and far better observers than myself, may, I therefore hope, be within the reach of all medical men.

I desire to take the present occasion of expressing my warm thanks to the many kind friends who have helped me. To those, whether of my own profession or without it—and they, I am thankful to say, are many—by whose example I have endeavoured to teach myself to be industrious and honest in my own labour, and to be generous in my estimation of the labour of others, naming them silently, I can offer to them no gratitude like the largeness of their gifts. To others, from whom in addition to this I have received especial assistance, I may have the pleasure of acknowledging my debts. From Dr. J. W. Ogle, at St. George's Hospital, I first received the idea of the probable value of the Ophthalmoscope in cerebral disease; from Dr. Hughlings Jackson I have received constant encouragement, and that liberal participation in the results of his works and thoughts which those who know him best will best understand; from Dr. Lockhart Clarke, Dr. Dickenson, and Mr. G. H. Lewes, I have received practical instruction in the microscopical dealing with tissues, and much valuable help besides; and

Mr. Swanzy and Mr. Hutchinson have enabled me to enrich my pages with the coloured drawings of the choked disk and of neuritis in lead poisoning, which were executed for them by Dr. Fitzgerald and Mr. Burgess. My colleague and friend Mr. Teale, again, has placed me under obligations which are as enduring as they are pleasant to be bound withal, for he has given himself, his experience, and his materials so generously and so continuously to me, that I can no longer define between that which is my own and that which really belongs to him. To Mr. Oglesby's patient and intelligent industry I am much indebted for the continuous observation and notes of many cases in which I was interested. Dr. Crichton Browne, the Medical Superintendent of the West Riding Asylum, has associated himself so thoroughly with my work, and, by his aid in many ways, especially in the supply and description of pathological specimens, has so fully and so unselfishly contributed to my undertakings, that I only fear, in giving him the thanks which are due to him, to make it too clear how disproportionate are his benefits to my accomplishments. At the North Riding Asylum, Dr. Christie has in like manner placed himself and his materials at my disposal on more than one occasion.

To those in whose writings I have found help and instruction I have made particular references in the course of my book.

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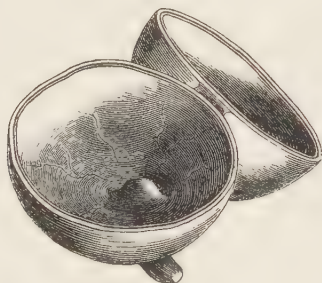
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DESCRIPTION OF FRONTISPIECE.

THE coloured plate represents the state described (*vide* page 54) as Ischæmia papillæ, and called by Gräfe the Stauungs papilla. It was drawn by Dr. FitzGerald from a case under Mr. Swanzy's care¹. The patient was a soldier who was admitted under Dr. Robinson for symptoms due to a sarcoma afterwards found in the centre of the cerebrum. The patient was blind and the pupils dilated. Mr. Swanzy noted in both eyes great tumefaction of the optic disk and tortuous and engorged retinal veins. The tumefaction did not extend far into the surrounding retina. After death the right eye was obtained for Mr. Swanzy by Mr. Baker, and the following drawing of the interior was made, showing the swelling of the disk.



There was no neuritis extending up the optic nerves behind the eyes.

¹ *Vide* 'Dublin Quarterly Journal of Medical Science,' February, 1871.

ON THE
USE OF THE OPHTHALMOSCOPE.

CHAPTER I.

INTRODUCTORY.

THOSE of us who find enough to fill their thoughts in time as it goes, who seldom look behind them, and who are consequently incapable of looking before them, such of my readers, if any such there be, have little idea of the rapid advance now being made in our knowledge of the nervous system. It is not many years since a physician, now living, a man of great acuteness, and one of the leading physicians in London, said to me:—‘They talk nonsense who pretend to localize disease within the encephalon; ingenious guesses you may make, but such guesses seldom prove to be worth much in the dead-house.’

Probably there was a little of the whim of a vigorous mind in this; but can we imagine that even a whimsical man, if he knew anything of the matter at all, could make such a speech to-day? Yet the speech was not absurd when it was made. How unfair such a saying would now be we have ample evidence in the writings of modern nervous pathologists; and no one, perhaps, would be more astonished than the speaker himself were I to remind him of his long-forgotten assertion.

The wonderful advance in our knowledge of the minute anatomy and pathology of the central nervous system has imbued a like spirit within clinical observers, and they, leaving the vain traditions of their forefathers, and adjusting

themselves less to preconceptions and more to things, are winning their way onward into the most cherished secrets of nature. Almost all this is due to the microscope—to the microscope and to that genuine temper of the observing mind which begot and is begotten by the microscope. Professor Rolleston, in his remarkable address on Physiology, read at the British Medical Association at their Oxford meeting, showed that the older anatomists were none the greater for their freedom from distracting ‘*microscopische spielereien* ;’ on the contrary, that they never reached the standard of accuracy in visible things which the microscope has since helped to establish. So it is in pathology: it would be idle in me to bring forward examples to show that morbid appearances without number, of a kind quite evident to the naked eye, were never described with any adequate care until the microscope raised the standard of care.

The minute precision of this and such instruments, so far from encouraging a narrowly curious habit of mind, has the very contrary effect. Not only was our knowledge of the diseases of the nervous system of a very meagre sort until the use of the microscope became general, but, I may add, that our method was even unworthy of our knowledge. Not only is the brain the most complex and least accessible part of the body, and therefore the last to benefit by the more vigorous and more philosophical mode of investigation, which may be said within the last few years to have changed the face of the medical art, but it is in the descriptions of the functions and of the disorders of the brain, that what has been called the metaphysical or transcendental habit of thought has most tenaciously held its ground. Where the order of phenomena is most complex and observation most difficult, there our theories most readily escape the test of experiment. Unchecked by direct reference to nature, theories which have a fair aspect, which are clothed in imposing language, and which are symmetrical and definite, there continue to command assent, although elsewhere discredited. No one would indeed now dream of referring the functions of the liver or of the heart

to an immaterial principle residing in or about these organs, yet many persons still cling to the opinion that the functions of the brain are something more than the movements and the relations of the cerebral tissues; and they not unnaturally therefore refer diseases of the encephalon to something more than the abnormal movements of its component parts.

We are tempted, for instance, to give a reality to such a disorder as epilepsy apart from the phenomena in which we say that it is seen. We are led to forget that molecular equilibrium may be disturbed to a greater or less degree in the brain as in any other aggregate; and instead of tracing out deviations from health, we satisfy ourselves with naming the morbid state as we see it in its fullest development, and having named it we try to hope that it is explained. We thus begin more or less consciously to use such a word as 'epilepsy' in the sense of a principle of causation, and to forget that it is merely a name given to a more or less definite group of irregular movements. Even in the writings of those who take a clearer view of the value of such names as 'epilepsy,' 'chorea,' and the like, we may often detect a tendency to use such words too much in a pictorial sense. A brilliant sketch of an epileptic state, for instance, is set before the reader, and is presented to him as a 'type' or standard, by which he is to regulate his conceptions of all similar states. Certain marked features are held to be necessary to the proper constitution of the 'type,' and all modes of irregularity of function not presenting such features are held to be what they please, but certainly not epilepsy. They must group themselves after a given fashion, and present certain given characters on pain of being neglected, or, at best, recorded as 'curiosities.' Yet it is in these slighter deviations from the normal order, in spasmodic neuralgias, local tremors, transient suspensions of the senses, and such minor indications of lessened tension and increasing instability, that we shall ultimately find the explanation of the more 'typical' forms of disorder. It is not by setting up opposition standards to the standard of health that we shall learn the modes of initiation of morbid changes, but rather by watching the outskirts of health itself.

Before we can comprehend extensive changes, we must familiarise ourselves with slighter ones, and so take with us the clue to the larger mystery. We shall, no doubt, continue to depict the extreme and complete manifestations of disorder for clinical ends, yet if we are to discover their origin, we shall have to desert this kind of synthesis for analysis. We must unravel groups of phenomena, and trace each element to its source. We must learn to have a less exclusive admiration for brilliant displays of disease, and to cultivate rather a perception of those many little various errors from healthy order by which Nature chiefly seeks to betray herself. A straw may show the way of the wind better than a falling tower. A habit of thus wakefully regarding the minutest variations of the normal state, and of verifying them accurately, is of inestimable value, and is quite the opposite of that other habit of setting up certain morbid standards or lay-figures to which all changes are to be referred. It cannot be too earnestly impressed upon our students that any new facts, however small, if well observed, may lead up, and probably will lead up, to some wider truth of scientific or even of immediately practical importance. But to compare individual instances of disease with conventional standards, is directly to discourage the observation of those lesser phenomena, and to teach the student rather to pare them off as far as possible until he can produce his case in trim with accepted models. The baneful influence of this method of case-taking is but too plain in all medical schools. Students are led to think that facts which seem to them to be accessory are not only unworthy of verification, but are even intrusive, and rather spoil the elegance of their case than otherwise.

I much doubt indeed whether such terms as 'epilepsy,' 'chorea,' &c., will prove ultimately to be valuable as names. Their signification will be found so indefinite as the study of temporary and chronometric failures of function advances, that I fully expect to see the groups which they profess to designate altogether broken up, and their elements grouped again under higher and more philosophical names having reference to other and wider affinities. We see this process in other

names, indeed, already going on. The name 'apoplexy,' for example, is retained in our nomenclature rather from habit than from any belief in its value; and the term 'inflammation' hangs on our lips by a very precarious tenure.

The way which is open to us for the discovery of the laws of change in nervous organs must be, to a great extent, therefore, a way of destruction. Nothing is so conducive to a right appreciation of the truth as a right appreciation of the error by which it is surrounded. The successful investigator must bring to test statements and conceptions which have been too long accepted on faith, habit, or good-nature. He must look boldly behind certain large words which are now too often the shelter of ignorance, and he must satisfy himself whether they have any definite value or not. When it is seen how much our current language really signifies, and when all technicalities, which took their rise in old and false methods, have been swept out of sight, we shall feel, perhaps, a little bare, but at any rate we shall have open field for our new researches. When we have stripped off all overgrowth of heavy verbiage, we shall see that there is no lack of facts, and in our endeavour to verify those which we think we have, we shall continually come across others which no ingenuity of our own could have led us to seek for, but which may turn out to be of the greatest practical value. Moreover, the steady pursuit of such a method strengthens in the observer that spirit of open-eyed sincerity which in the man of science answers to the catholic sympathy of the greatest artists, and is the true magistry.

It is therefore with great anticipations not only of a direct increase of knowledge, but also of a great purification of method and of speech, that I now see the ophthalmoscope, another arm of precision, brought to bear upon nervous diseases; an instrument requiring minute accuracy in the use, and revealing modes of nervous change during life which before could be known only after death and in their results. I regard the application of the ophthalmoscope, not to the diagnosis only, but also to the investigation of modes of nervous change, as of very happy augury. It will, like the microscope, not only teach us to see the new things which it

exhibits itself, but it will train our eyes to see many more new things which before we had overlooked.

My readers well know the marvellous change which this instrument has produced in the knowledge and method of the oculist. Not only has it cleared up for him many doubts, and has enabled him to recognise certain pathological states which before were beyond his reach, but the new habits of accuracy which it has encouraged are very evident also in recent work in those departments of ophthalmic practice where the ophthalmoscope is less needed. Recent inquiries, for example, into the disorders of accommodation and refraction, and of the muscular action of the orbit, appear to me to have been conducted in a genuinely scientific spirit, and have led to results whose bearing upon more general laws of nervo-muscular life may turn out to be most important¹. Whatever, then, may prove to be the practical value of the ophthalmoscope in detecting disease of the brain or spinal cord, it has for me this great charm—that its use must favour a spirit of industrious and accurate observation, and must favour also that wholesome disposition of mind which welcomes any facts, however far away they may seem to be from traditional doctrines or dignified theories. I can scarcely suppose that the ophthalmoscope will, in the hands of the physician, ever rank in usefulness with the stethoscope. I confidently believe, however, that as the invention of the stethoscope has been of incalculable advantage to us, not directly only, by revealing changes of tissue during life, which previously could be but roughly guessed at, but also indirectly, by encouraging the study of diseases of the chest; so the ophthalmoscope will help us, not only by the facts it directly reveals, but by stimulating work in the direction of nervous diseases. Nor must it be forgotten that by means of the ophthalmoscope we are for the first time permitted to see the commencement and progress of change in the life of nervous tissue, and to ascertain the modes and times of such change.

This is not a slight matter; and if to all these considerations

¹ Cf. e.g. Gräfe, 'Klinische Analyse der Motilitäts-störungen d. Auges,' and the many treatises which have followed it.

I add, as I shall presently show, that the ophthalmoscope is even already of much use in diagnosis, I shall have made it clear that this instrument must be in the hands of every physician who wishes to speak with authority on the subject of diseases of the nervous system. The great drawback to the rapid introduction of new instruments is the labour required in learning their use. Thus it is that many useful aids to diagnosis—the laryngoscope, the endoscope, the sphygmograph—have a kind of alacrity in sinking out of notice. Every medical school is now, however, bound to teach its students the use of the ophthalmoscope as carefully as the use of the stethoscope is taught. But it is not easy for physicians who have left the schools, and are engaged in practice, to take up a new instrument which requires much skill in the using. I can assure my readers, however, that a few hours spared for this work are very well spent. The new glimpse thus gained of a number of obscure and difficult diseases adds greatly to the interest of study; and I hope to show that the ophthalmic signs of intracranial disease are so many and so important, that the reader will probably agree with me that no records of nervous diseases can henceforth be called complete which do not contain an account of the ophthalmoscopic appearances.

It has long been known that indications of changes in the nervous system were to be found in the eye. Motor aberrations, such as contraction or dilatation of one or both pupils, squints, ataxy of the ocular muscles, and imperfect accommodation; disorders of vision, such as photophobia, diplopia, hemiopia, and even amaurosis, have all been recognised as occurring in connection with central disease. It was not possible, however, until the discovery of the ophthalmoscope by Helmholtz to attach any other than a very loose meaning to the word ‘amaurosis’². Suspension of the visual functions is often due to other causes than to disease of the optic nerve or retina, and it is probable that some cases of so-called amaurosis are actually due rather to troubles of accommodation

² Witness the often ill-quoted epigram of Walter, ‘Amaurosis sei jener Zustand, wo der Kranke nichts sieht, und auch der Arzt nichts.’

than to any deficient power in the nerve of sight. A minute study of the disorders of motility in and about the eye is quite as important as a study of the variations of the optic nerve itself. A slight droop of the upper eyelid, and an equally slight deviation of the axis of the eye, will reveal the existence of a meningitis to the physician who had previously hoped that he was dealing only with a fever. I am unwillingly obliged, however, now wholly to pass by other symptomatic affections of the eye, in order to give exclusive attention to the alterations of the optic nerve and retina, considered mainly in their relation to cerebro-spinal disease.

It is but very recently that the profession has been made aware that the interior of the eye presents any visible indications of the disorders of the nervous system, nor can we say even yet that the great importance of these indications is generally understood.

Another great drawback to the full appreciation of such facts is the unlucky division of cases between the physician and the ophthalmic surgeon. If the disturbance of sight be that which most affects the patient, he goes the round of the ophthalmic hospitals; if, on the contrary, the disturbance of the nervo-muscular functions be uppermost, he falls under the care of physicians, who are naturally prone to overlook any changes of the inner eye. As marked changes may occur at the back of the eye with slight or with no disorder of the visual function, it is not surprising that the physician should overlook one half of the facts, and it as naturally happens, on the other side, that the surgeon's attention is equally limited. While the present absurd division of the profession into operators and non-operators continues, we must be content to urge upon those physicians who take an interest in nervous diseases to frequent the ophthalmic hospitals, where a wealth of material awaits them, of which they have little conception. I am able to assure my medical brethren that they will receive a warm welcome from their surgical allies, who, in their turn, are much interested in the relations of eye affections to more general diseases. Indeed, physicians have little idea how 'medical' are the 'Ophthalmic Hospital Reports' and the

'Ophthalmic Review;' and to the medical work of ophthalmic surgeons like Mr. Hulke, Mr. Hutchinson, and others in England, and like Gräfe, Sichel, Liebreich, or Desmarres abroad, physicians are already deeply indebted. I wish I could say that the physicians showed a greater sense of their obligations. The number of physicians who are working with the ophthalmoscope in England may, I believe, be counted upon the fingers of one hand. If I may judge from the publications of Galezowski and Bouchut, it would seem that the same reproach cannot attach to our Continental neighbours, who will, therefore, unless we bestir ourselves, make this large field of observation more especially their own.

Dr. John Ogle was the first physician who called my attention to the probable results of ophthalmoscopic examination in cases of cerebral disease; and he published a paper on that subject more than ten years ago in the 'Medical Times.' Dr. Ogle then impressed upon our notice the very close relations which exist between the cerebral and the intraocular circulation, and he urged that the beautiful vascular structure of the posterior parts of the eye might serve in its variations as an index to the vascular condition of the intracranial organs.

That 'dim suffusions and cecities the most serene' do often visit the orbs of those suffering from cerebral disease is, as I have said, no new discovery. I had made a list of references and quotations from a long series of medical authors anterior to Gräfe who notice this connection, and I had intended to publish the list here; it became, however, a very long one, and, after all, that which we have to do at present is not so much to establish the connection between cerebral and visual disorders, as to establish, with the ophthalmoscope and the microscope, the modes and times of these relations for purposes of insight into the ways of nervous disease in general. My object is not so much to prove the common concurrence of the two sets of symptoms, as to discover the manner of it and its calculable value in pathology and in diagnosis.

But it would be unfair to forget that something had been done in interpreting the connection between amaurosis and brain disease before the ophthalmoscope had been even thought

of. It had not only been pointed out that atrophy of the optic nerves often followed scrofulous disease of the base of the brain and other such cerebral affections, but also a few microscopic investigations had been made by pathologists in such cases³. In the præ-ophthalmoscopic period, however, amaurosis from cerebral causes was generally put down to a simple paralysis of the optic nerve, with some wasting, perhaps, as a remote consequence. Even those who are best familiar with the ophthalmoscope will find it difficult to realise the fact that fifteen years ago a descending neuritis was never dreamt of, and was first revealed by the mirror to Sichel, Gräfe, Liebreich, and the other earlier ophthalmic investigators.

The idea of an ophthalmoscope was suggested by Cumming more than twenty years ago. Such an instrument was invented afterwards by Helmholtz, to whom modern science owes so much, and was described by him in his 'Beschreibung eines Augenspiegels,' published at Berlin in 1851. The invention created but little interest at the time, and Mr. Spencer Wells⁴ was among the first in England to insist upon the great value of the ophthalmoscope in diseases of the eye. It is difficult to say to whom we owe the first important and careful observations of the modes of consecutive disease of the optic nerves. Sichel and Gräfe were perhaps the principal workers at first in this new field of observation, and the well-known essay of the latter, 'Ueber Complication von Sehnerven Entzündung mit Gehirnkrankheiten,' in the 'Archiv. für Ophth.' Band VII. Abtheilung ii. S. 58, published in 1860, drew general attention to the great importance of the subject. The study of these morbid changes of the disk and vessels was thenceforth vigorously prosecuted by Sämisch, Liebreich, Schweigger, Hutchinson, Carter, and many others.

Were I now about to treat of paralysis of the optic nerve merely as a symptom, merely as one deviation from the normal among the many which constitute the several combinations

³ Vide Jüngken, 'Lehre v. den Augenkrankheiten,' Berlin, 1832, and such monographs as that of Davis on Hydrocephalus.

⁴ 'Medical Times' for September 10, 1853.

significant of certain and several cerebral lesions, then it might be my duty to include in one survey, not the optic nerve alone, but the other nerves of the orbit also. Or, if I conceived, again, that the optic nerves were attacked on any transcendental grounds—on grounds, say, of the intimate association of the visual function with the higher muscular co-ordinations—then it would be my duty carefully to compare the failures of the special nerve of sight with the failures of the special nerve of hearing, of taste, or of smell, and to include all the nerves of the special senses in one survey. A strong distrust of transcendent reasons, however, combined with, or rather consisting in, a strong trust in anatomy, together with some further confidence, justifiable or otherwise, in my own researches, leads me to believe that the sufferings of the optic nerve are due entirely to the peculiarities of its own structure and attachments—to its rich vascularity, its large share of connective tissue, and its extensive relations with the parts at the base of the encephalon—and not in any way to its special attitude as a sense, not even in such disease as locomotor ataxy. I must set aside, then, as foreign to my chief purpose, all discussion of the very interesting affections of the orbital muscles—a discussion which is full of interest to the student of palsies, but which I must leave for another occasion⁵. The affections of the optic nerve and retina are so various and so important, and shed so much light upon both pathology and diagnosis, and they are concurrent also with so many lesions of distant organs, that I shall bestow enough of my tediousness on the reader in dealing with them alone.

I shall, in the first instance, describe the anatomy of the optic nerve and retina so far as may be needful to give us a true knowledge of the healthy standard, and in this description I shall assume a good deal as known that would otherwise cumber the page. I shall, in the next place, endeavour to trace the mode and time of variations from this standard, beginning with the simplest and earliest. Having done that, I shall be at liberty to take the various diseases in turn,

⁵ I hope hereafter to follow up this volume by another essay on the disturbances of motion and nutrition in the eyeball.

encephalic, spinal, renal, and others with which optic changes are associated, and to describe, as nearly as I can, the way in which such associations take place.

Before entering upon these chapters, however, I propose to make a few remarks upon the manner of investigating the states of the optic nerve and retina.

CHAPTER II.

ON THE EXAMINATION OF THE EYE.

I DO not purpose to give here such a description of the methods of ophthalmic examination as we look for in comprehensive treatises on ophthalmology, but only to give a few brief hints, addressed more especially to medical observers.

First, as regards the choice of instruments. The portable instruments, with a glass mirror and one convex lens, are very defective. The mirrors should be made of metal, as in the glass mirrors we have a double reflection at the wide sight-hole, one from each of the two surfaces of the glass; we avoid this in the metal mirrors by paring down the metal very thin near the centre, and then piercing a sight-hole which ought to be very small. Moreover, one two-inch convex object-glass is insufficient; a second of about three and a half or four inches is quite necessary; and a concave lens is indispensable for the direct examination. The observer should therefore get a metal concave mirror of about ten or twelve inches focus¹, with a clip behind it to hold ocular glasses; he should have, also, two

¹ Many mirrors now sold have no focus at all. It is impossible with such mirrors to regulate the circle of dispersion upon the retina with any accuracy. Mr. Carter has recently shown that the mirrors in common use are too large. He considers that a mirror ought not to exceed a shilling in size (vid. 'Lancet,' Dec. 24, 1870).

convex object-glasses, of two and four inches focus respectively; and, finally, a concave lens for the direct examination. If his own sight be defective, he must have in addition an appropriate correcting ocular lens. Instead of having two convex objectives, he may have one of two inches value, and supplement it by a small magnifying ocular lens; but it is desirable to have two convex object-glasses, as in a short-sighted eye a weak convex lens is needed, and a strong one in presbyopia. My readers are perhaps aware that there are two methods of examining the eye—the direct and the indirect method. The direct method gives a very large and distinct view of minute parts, but the field of observation is therefore small; it is indispensable, however, in all careful investigations, and it is the only method by which we can form an accurate estimate of variations in colour. The direct method, as its name signifies, is the method of looking directly at the retina itself. In the direct method we act precisely as we do when we look at a picture through a common magnifying lens, the crystalline lens of the eye being the magnifier, and the retina the picture.

Every one knows that in looking in this way at a picture or other object, the image seen is deceptive in this, that it appears to be not in the actual plane of the object itself, but in another plane; which fact is expressed scientifically by calling the image seen not a real, but a virtual image. To carry out this mode of examination, we have to approach our own eye, armed with the mirror, very close to the patient's eye, and it is desirable, therefore, to examine the patient's right eye with our right eye, and his left eye with our left, to avoid rubbing of noses, and also to leave the patient free to adjust his vision for the distance or in the direction we require. The observer must close the eye not in use. A close approach is necessary in the direct method, partly on account of the small aperture through which we have to look, and partly on account of the action of the refracting media. Our eyes are primarily constructed to bring parallel rays to a focus upon our retina, and we have in addition an accommodating power in the lens to enable us to condense divergent rays also, so as to focus these

upon our retina. But we have no dispersing accommodation to enable us to retard convergence of rays, and to delay their uniting until they reach the retina. So, if we have to deal with convergent rays, these will meet at a point anterior to the retina, unless we can open them out by some artificial means. In the direct examination we very often have to deal with converging rays, or with rays that will be seen the better for a little more dispersion, and for this end we interpose the concave lens. We thus make the rays divergent, in which state they are easily dealt with by our accommodation, and focused upon our retina so as to form a picture or image.

In the indirect method, as its name likewise implies, we no longer look directly at the retina, but we form an image of the retina outside the eye, and look at that. As this image is actually formed in the plane in which we see it, it is no longer to be called a virtual image, as in the last case, but a real one, and by looking at this real image of the retina we indirectly see the retina itself. The image is made by the interposition of a bi-convex lens in addition to the crystalline, and is therefore inverted. The rays falling from the mirror are condensed by the lens, and directed into the eye; then the rays which spring divergent from the reflecting surface of the retina are recondensed by the crystalline lens, and emerge as convergent pencils. These pencils are rendered still more convergent by passing again through the artificial lens, so that they are brought to a focus in front of it and between it and the observer. If the lens be of two-inch focus, these rays will be united, and will form a picture two inches in front of the lens. At this picture, and not at the patient's eye, must the observer look, and after a little practice the observer will learn insensibly to accommodate his vision for this point. All observers will do well to use a binocular or stereoscopic instrument at some time in their lives. This instrument is not by any means necessary for common use, but it is well to make ourselves so familiar with the perspective view of the parts of the inner eye, that we may readily interpret the appearances of the flat picture. For example, any cupping at the back of the eye can only be represented as a flat picture

by means of a monocular instrument, but if our memory is familiar with the perspective view given us by means of a binocular instrument, we more readily infer the dimensions of the cup in depth. It is often very important, as we shall see, to distinguish the cupping of amaurotic atrophy from that of a commencing glaucoma. The binocular instrument is used for the indirect method of examination, for the examination, that is, of the real inverted image.

Many practitioners shrink from the use of the ophthalmoscope, because they believe that a troublesome arrangement of light and darkness is a necessary condition. This notion is encouraged by the especial arrangement known to be in use at Moorfields and elsewhere. Such management is less necessary, however, than we commonly imagine. A well-darkened room, and a bright and convenient lamp, are very important at ophthalmic hospitals, because the observations taken there are to be of standard value, and are, moreover, to be made as easy as possible for students and unpractised gazers. But after some practice, these arrangements are found to be less needful than is commonly supposed. There are very few houses in which I find it difficult to make an ophthalmoscopic examination. If the blinds are drawn down, a reflection may be thrown into the eye from almost any gas pendant or bracket; or, in default of gas, a common candle may be made to give light enough to show whether there be changes at the back of the eye or not, and in the former case to show of what kind the changes are. For minute investigations in our consulting-rooms, a curtain or shutter and a good lamp should be provided. For patients confined to bed, either a candle may be used—a wax candle if possible—or perhaps a small lantern containing a bull's-eye lens, after a pattern once suggested to me by Mr. Carter. I am rather disposed, however, to dislike light from a distant concentrating lens, as it is difficult to manage. M. Galezowski and other ophthalmologists use a kind of instrument which is adapted to daylight examinations. It consists of two tubes, blackened within; of these, the lower and wider is fitted with a pad to the orbit, and the upper and narrower one travels within it, carrying

the lens at its lower, and the mirror at its upper end. It is unnecessary, however, and I think inconvenient, to have the mirror fixed. The tubes may be used quite well with a detached mirror, held as usual in the right hand. But for my own part I seldom now use Galezowski's instrument; for a little dexterity will enable any one to ascertain, even in daylight, whether the disks and vessels are normal or not, and within certain limits, indeed, to ascertain the kind of morbid change, if any there be. Where, as in a hospital ward, the room cannot be darkened, and the patient is in bed, and these are the most unfavourable circumstances, I can generally succeed by placing a wax candle or a small paraffin lamp in the proper position, and by shading the orbit at the side with the hand which holds the lens, and in front with my body, to obtain a useful view of the back of the eye. These rough expedients often give us valuable knowledge which would otherwise be entirely lost.

Dr. Beale has constructed an ophthalmoscope which, like that of Hasner, carries the mirror, the lens, and the lamp in one. I have a great dislike to fixed parts in any ophthalmoscope, and the more the parts fixed the more troublesome I find it to manipulate them so as to throw aside teasing reflections, to vary the positions of the lens, and to hunt over the whole retina. Moreover, I like to change my lenses at will, or to pass from the indirect to the direct examination, all of which is impossible in fixed instruments. On the other hand, I fail to see any advantage in fixing the lamp, even when the lens must be fixed to a tube².

The use of the separated mirror and lens for indirect examinations cannot be taught by books, but may be taught in five minutes by a demonstrator. The lamp is now placed

² Since this was written I have made a useful daylight instrument in the following way. Instead of fitting a padded tube to the orbit, I have a black silk curtain which is attached all round the rim of the lens, and thence falls round the orbital region of the patient. The lens is fixed to a short blackened tube, within which the curtain is retracted when the instrument is not in use. The mirror, unattached when in use, is screwed as a lid upon the tube when not in use. With this instrument movement is easy in every direction. The patient is examined in bed or upon a couch.

on the left of the patient only, and the operator takes the mirror in his right hand, and the lens between the first finger and the thumb of his left hand. He then rests the second and third fingers of this hand on the patient's forehead, so as to steady the lens, and moves it to and fro until he finds the focus. The fourth finger remains at liberty, to raise the eyelid if necessary. In varying his lenses, he will remember that, generally speaking, the size of the field depends on the degree of the enlargement and on the width of the pupil; the greater the enlargement the smaller of course is the field, and the reverse. When, as in the direct examination, we can approach the pupil, we obtain, as I have said, a larger field. In the indirect examination it is best to arrange the condensing lens so as to bring its focus upon the iris, the rays then cross over and form a circle of dispersion upon the retina. A plane mirror is easier for a direct examination than a concave one, as it catches the light better; but this difficulty is soon got over. The observer can only learn by frequent practice how to escape teasing reflections, and how to hunt over the retina; but, if he has no personal disadvantages to contend with, he will soon master all this. At the commencement he will do best to examine an eye under atropine, the use of which must afterwards be restricted. A practical observer will generally try to do without atropine. If atropine be used constantly, patients soon become annoyed by the very unpleasant effect of paralysing the accommodation; sometimes they will declare that their sight has been permanently injured by it, especially if their amaurosis be progressive. Moreover, unless very strong solutions be used, it is a certain waste of time and trouble to treat the eye with atropine, and to wait until the pupil be dilated. When, therefore, the observer has become skilful enough to hold the mirror still, and to throw the beam steadily upon the blind optic disk, while the patient accommodates for a distant point, he will find that by so doing he no longer keeps up contraction in the pupil, and he is enabled to see well enough into the

state of the optic nerve and chief vessels. In the great majority of cases this is sufficient for the physician.

Should there be extensive change, however, in the retina, rendering a large field of view necessary, as in retinitis, or should it be intended to use the case for class demonstrations, then it will be found needful to dilate the pupil artificially. Such cases are the exceptions. Atropine will also be needed for cases where the patient is unmanageable, or when the pupil is closely contracted, as it often is in such diseases as locomotor ataxy and general paralysis. On no account should we drop atropine into the eyes of children; they never forget it, and all hopes of ever seeing their optic disks is henceforth lost. If children are gently and cautiously handled, the disks may be seen in the large majority of instances. Let the mother hold the child in her arms in a natural, easy position, with its back to the lamp, and then let an assistant dangle a bunch of keys in the direction in which the visual axis should be. The disk will come quickly into view, and must be caught flying. I not infrequently find that babies are almost or altogether blind from atrophy of the optic nerves, without the mother having any suspicion of it, or she may perhaps describe the child as late in 'taking notice.' We should therefore never omit the attempt to examine, even if the child be fretful. It has often happened to me to see a fretful child charmed at once into silence when the beam has been thrown into its eye. The wonderful bright thing may fascinate it, and make it forget its woes for an instant; if so, let not the golden moment slip away, for it does not return. Suckling infants should be put to the breast; while the mother gives suck and caresses the child, the observations may in the majority of cases be easily made.

I have stated in the journals on several occasions, that it is of great importance to ascertain the boundaries of the field of vision. The mirror, without this help, may at times be insufficient to establish a diagnosis: for example, the distinction between anæmia and atrophy of the first degree may be out of our reach if we look to the mirror

alone. The elegant and ingenious, and, as it would seem, very effective instrument exhibited by Mr. Adams at the meeting of the British Medical Association at Oxford in 1868, bids fair, in large institutions, to supersede other means of mapping the field. I presume Mr. Adams has slips of paper ruled to correspond with his glass hemisphere, and that he transfers his tracings to them for permanent record³. I have been in the habit of using the old plan as modified by Mr. Teale, which answers quite well enough. He hangs a light black board against the wall from a pulley, in such a manner that it may be raised or lowered so as to bring the bull's-eye opposite to the patient's centre of vision. The board is ruled, like a target, with concentric circles. When the observation is to be made, a head-rest in the form of a crutch, and measuring eight inches in length, is placed with its pad against the patient's forehead, just above the orbit, and its point is placed perpendicularly upon the target. A white disk of paper at the end of a rod is then moved about upon the field in such a way as to test the range of the patient's vision in all directions, while his eye is opposite and constantly directed to the centre. As the disk is shifted from place to place, a plus or cipher mark with a piece of chalk registers the result upon the board. After the completion of this process, the map of the field is readily transferred with accuracy to a slip of paper ruled on a smaller scale to correspond with the target; this slip is filed with the day's notes of the case. The method gives trustworthy results, and it seems perhaps all that need in a general way be desired. It will hereafter appear how important it is that records of this kind should be taken from time to time in cases of atrophy of the optic nerves. It will thus be found that many patients having distinct optic atrophy or neuritis—and who may appear at the same time to have good vision—

³ Dr. Jeffries of Massachusetts has kindly forwarded to me the description of his records of the visual field. It differs little from Mr. Teale's plan, the most important difference being that the black board and the recording slips are divided into squares of known proportions.

have good central vision only, and have in reality lost a large slice of the lateral portion of their visual field.

In cases of intracranial tumour indeed, when the tumour is at the base, the state of the visual field is often of great importance to us in pointing out whether the nerves be attacked in their course. For if the tumour is seated upon one nerve before the chiasma the whole of the field will be destroyed in one eye, whereas if it cuts one nerve or tract behind the chiasma the right or the left halves of the field, as the case may be, will be destroyed in both eyes.

CHAPTER III.

ON THE ASPECT, STRUCTURE, AND CONNECTIONS OF THE NORMAL OPTIC NERVE AND RETINA.

THE present chapter need not be a long one; but a short one is necessary, not only on account of some details which need description, but also because I desire to lay practical stress upon the true method of investigating disease—the method, that is, of investigating minute and early deviations from health rather than great examples of mischief. There is no essential difference between Pathology and Physiology, though we think and speak continually as if these two studies dealt with distinct classes of phenomena. Pathology is but the shady side of physiology; their district, though seen under different lights and in various aspects, is one and the same; nor can any one really know one aspect without knowing the other also. Whether, then, disease be slow or rapid departure from the paths of normal development, this departure must still follow the certain laws of tissue change, if not the particular processes of health; as the reader and I, if we stray from the Queen's highway do still walk with legs, and do not, in becoming trespassers, cease thereby to be anthropological. So tissues which yield to temptation and, as Mr. Matthew Arnold would say, strive no longer to realize their best selves, do not thereby cease to concern the physiologist, but on the contrary, by the very mode of their errors, do more fully demonstrate their physiological capacities. The true view of life must comprehend not only the normal or most complex tissues, but

also the multitudinous forms into which these may change by loss of complexity: the false view regards physiology or normal states of tissue as one thing, and pathology or abnormal states of tissue as another. Sailors no doubt regard storms and eclipses as highly pathological, and feel aggrieved at the philosophical indifferentism of the astronomer at home; but they are wrong, and so are we when we forget that no tissue can be called normal or abnormal except in relation to particular parts, and that what is normal in one place becomes abnormal if found in another. Our duty then is, first of all to familiarize ourselves with the general laws or modes of tissue growth as we see them throughout the microcosm or the macrocosm, and secondly to learn the special modes of tissue growth for each particular part.

Having done this we shall be in a position to call the tissue we find in any place normal or abnormal, not on account of certain features of its own taken apart, but on account of its congruity or incongruity with the characters of a particular creature, a particular organ, or a particular part. To determine what is abnormal then in the eye, we must first carefully ascertain what are the peculiar modes and relations of tissue growth in the constituent parts of the retina and optic nerve, and their connections, and how far the variations of these can be recognised directly or indirectly by means of the mirror, or by the scalpel after death. Many parts of such a description may of course be rapidly dealt with as they are inessential or familiar to us all, and this the more as after some hesitation I have determined to confine myself in this treatise to a consideration of the parts connected with the optic nerves only. The other parts of the eye, however, and the muscles of the orbit present many changes of a most interesting kind in themselves, and which have important relations with disease elsewhere: thus the affections of the mucous covering of the eye should be studied in connection with the skin and other tegumentary surfaces, and the affections of the fibrous tissues with fibrous tissues elsewhere¹;

¹ The cornea has recently been proved by Schweigger Seidel to have affinity with the connective tissues.

while the study of the muscular aberrations in the orbit present many most interesting problems and illustrations to the student of palsies and of motor conditions generally. To do all this well would need, I find, a book much larger than I ought now to write; in confining myself to the nervous parts of vision, and describing and explaining their changes, I shall have a task quite large enough for my powers.

Beginning then from before, backwards, we find a delicate coat, consisting in great part of nervous matter, which lines the greater part of the cavity of the eyeball. This coat or layer—the retina—is very transparent, so transparent indeed in fair people as to be invisible under ordinary circumstances; it is nevertheless a structure of very high complexity, and one which presents many interesting variations in different animals. It is incorrect to speak of the retina merely as an expansion of the optic nerve, as is too frequently done; and it is incorrect not only in anatomy but also in pathology, for the retina shows a good deal of independence of the optic nerve in its liabilities to disease as well as in its normal structure and its means of nutrition.

The accurate and valuable researches of Schultze into the minute anatomy of the retina, prepared with osmic acid, have added much to our knowledge of one of the most interesting organs of the body². It is very satisfactory to be able to add that our countryman Mr. Hulke has not allowed all the honour of these recent investigations to go to our Continental brethren³. Among former observers we owe most, perhaps, to Müller, Bowman, and Kölliker. It would of course be beyond my purpose to enter now upon any description of the delicate tissues of the retina; I must

² M. Schultze, '*Zur Anatomie und Physiologie der Retina*,' Bonn, 1866; vid. et '*Arch. für Mikroskop. Anat.*,' Band III. S. 215, 372, 404.

³ J. W. Hulke, '*The Anatomy of the Retina in Amphibia and Reptiles*,' in Royal Soc. Archives, 1864. '*On the Anatomy of the Fovea Centralis of the Human Retina*,' Phil. Trans. vol. 157, Part 1, p. 109. '*On the Retina of Amphibia and Reptiles*,' Journal of Anat. and Phys., Nov. 1866, p. 94. '*Notes on the Anatomy of the Retina of the Porpoise*,' ibid. Nov. 1867, p. 19; and '*On the Bloodvessels of the Retina of the Hedgehog*,' Phil. Trans. 1868.

content myself with a short indication of its structure and relations.

The *tunica nervea* is continuous with the optic nerve and lies within the globe of the eye upon the vascular coat or choroid. When looked for with the mirror in the healthy eye, it is so transparent as almost to escape observation except in very dark people, and the red choroid which shines through receives only a slight gray tint from the overlying retina. This is true, however, of the layers of the retina only; its vascular part, on the other hand, is beautifully distinct, and has to be carefully watched by the physician, as we shall see hereafter. An artery (*arteria centralis retinae*) enters the retina, and two veins (*venae centrales*) leave it near the centre of the optic disk, passing between the bundles of fibres in the optic nerve, and so reaching the ophthalmic artery and the ophthalmic vein respectively⁴. The artery generally bifurcates into a superior and an inferior branch after it has issued from the disk, or at any rate after it has come so far forward that the bifurcation is to be seen through the transparent nerve bundles. Immediately afterwards it divides into four or five main branches, which again distribute themselves to the retina on all sides, subdividing until they become capillary, and so return into the venules⁵. The venules gather themselves in the contrary directions, and assemble in the two branches of the central vein; these two branches, however, do not meet on the hither side of the sclerotic, but dive separately into the disk, generally at a small distance from the artery. Within the optic nerve trunk they quickly unite and form one trunk, which ends in the ophthalmic vein. The central vein approaches, and for a while closely accompanies the artery, but is never enclosed in the same sheath with it. One remarkable fact in the vascular distribution of the retina must not be overlooked, and that is the deviation

⁴ It would be a waste of time and space to describe minutely the distribution of the vessels, as they vary extremely in numbers and direction in different people. The point of their entrance and exit is also various: in a person I once saw the point was outside the rim of the disk.

⁵ In many animals there is a curious sinus around the *ora serrata* called the *circulus venosus*.

of the vessels from the region of the yellow spot. This, in my experience, is invariable. The branches on the external side which turn towards this spot, bend and form arches round it: no vessels of more than capillary size approach it, so that it is thus left unimpeded for clearest vision. The observer must bear in mind that the fovea centralis to the ophthalmoscope appears indeed to be extravascular. It is not generally difficult to distinguish between the larger arterial and venous branches in healthy retinas. The arterial branches are smaller, straighter, and of a paler or rose colour, while their transparency gives them the appearance of a double outline⁶. The arterial branches, again, are more superficial than the venous, and may often be seen to cross them; indeed, at times when the veins are full, the overlying arteries may appear even to strangulate them. In retinal hyperæmia, however, the distinction between arterial and venous branches is soon lost. These remarks must be understood to apply to the main branches only, but the smaller branches can easily be traced up to these. Besides the central vessels and their branches there is also a variable number of small arterial and venous twigs to be seen on the papilla. These branch and anastomose variously with the vessels of the retina and choroid, and thus form a vascular connection between the ciliary and retinal blood-vessels. (Leber, quoted by Stellwag.) These small vessels are much increased in number and in size during states of hyperæmia. The retinal coat may be traced with ease after death, as its transparency becomes clouded and a white filmy tunic is then to be seen extending from the disk to one-eighth of an inch behind the cornea, becoming thinner as it advances, and ending in a finely serrated edge called the *ora serrata*. The thinnest portion of the retina, however, is at the before-mentioned spot of Soemmerring, which spot is almost, though not exactly, in the axis of the eye. The optic disk—the blind spot—is slightly to the inside of the yellow spot, and is insusceptible of light. The constituent tissues of the retina are, as I have said, very complex, and need not

⁶ This appearance is now said to be wholly or partly due to the different refractions of the arteries and veins.

here be described. To two of its constituent tissues, however, I think it is desirable to refer—to the connective tissue framework and to the nervous layer. Without discussing the genetic relationship of the more specialized layers of the retina to connective tissue, I may say that we have known, since the publication of H. Müller's researches, that there is a distinct framework of this bindweb consisting of stouter radial and more delicate trabecular parts which comprise and support the more highly specialized structures. This fact is of great moment in estimating the probable character of inflammatory changes in the retina, and in explaining the occurrence in it of gliomatous and other tumours. The internal or nervous layer of the retina consists of two parts: the first or outer is a stratum of nerve cells and granules, and the second or inner is a stratum formed from the fibres of the optic nerve, which on leaving the optic disk lose their sheath, so that their central part or axis only is carried forwards. These axial fibres soon run into a continuous film, forming at first a delicate web with narrow elongated meshes. There is no fibrillar nervous matter, however, at the yellow spot.

With the convergence of the vessels we reach the papilla, or optic disk, a little part of great importance in our present inquiry, and which I shall therefore describe somewhat more at large. This disk, the entrance of the optic nerve, is the most striking object at the back of the eye. It is a small round or oval spot, of a delicate rosy or creamy white tint, contrasting with the pinkness of the surrounding choroid and with the retinal arteries and veins which traverse in order to dip into the optic nerve bundle near the centre of it. The margins of the disk in the healthy eye are even and sufficiently well defined; though in disease, as we shall see hereafter, its contour may become much sharper or much dimmer. The form of the disk is generally an oval with the longer diameter vertical; the vertical diameter, however, exceeds the transverse by a very small difference, and in many eyes the disk is round. I have in a few cases seen the disk transversely oval; in one of these the variation was only apparent, and was due to the defect of the cornea

known as astigmatism; in other cases it seemed to be an unimportant peculiarity, though I have twice seen it in cases of atrophy. The size of the disk is actually about 0.75 lines in its transverse diameter, and about 0.7 lines in its vertical, according to the measurements of Jäger⁷ and H. Müller⁸ taken in full-sized eyes; in many persons it is probably less than this. The apparent size, and also the colours of the disk, vary with the method of observation, whether direct or indirect, and again of course with the power of the lenses used in examination: with an ordinary two and a quarter inch convex lens the inverted image appears to be about the size of a small split pea, and with a four inch convex lens of about the size of a groat. When magnified to this size, as it ought to be in all important cases, or when examined by the direct method, its texture becomes more and more definite, its edges shade off more into the fundus, and the degrees of its vascularity are far more easy to determine. Finally, the apparent size of the papilla is changed by the conditions of the refracting parts of the eye.

The margin of the disk is sometimes surrounded by a crescent or ring, which may be white or black. The white ring may mislead an unwary observer into the supposition of a cupped disk, a staphyloma, or an atrophy. The variation I mean is however congenital, and is explained in two ways: one explanation is that the choroidal ring through which the optic nerve trunk passes into the interior of the eye may be a little larger in diameter than the trunk, in which case more or less of the brilliant bluish white sclerotic coat would be exposed to view through the transparent nerve fibres, and would appear as a segmental or circular collar around the disk; the other explanation is, that the fibres of the internal neurilemma of the optic nerve are not arrested at its entrance into the globe, but are prolonged up to the papilla, in which case there would be a white ring or crescent round the latter,

⁷ 'Ergebnisse der Untersuchung der Menschlichen Auges mit dem Augenspiegel.' Wien, 1852, pp. 8-10.

⁸ 'Anat. Phys. Untersuchungen über die Retina.' Leipzig, 1856, pp. 80-82; vid. et Gräfe's Archiv. N. 2, Müller and Liebreich.

not interfering with vision. I am disposed to think this second explanation the more probable, as a faint white ring round the disk may always be detected in the erect image, and indeed in some abnormal eyes the neurilemma is on the contrary lost too soon, and there is a want of substance about the disk which may simulate excavation. I have seen one or two such cases. A very little practice will make it easy to the observer to recognise an excess of neurilemma. He will see that there is no glaucomatous excavation, that the whiteness does not encroach upon the disk, and that the vessels do not dip under it as they would dip under the edge of a cup. If the circle or, as is more common, the crescent be black, it is due to a deposit of pigment in the choroid around the disk, and is of no ill meaning. I believe that it is sometimes stated to be peculiar to people of advanced years, but I have certainly seen it occur, and have sketched it as existing, in the eye of children. It is never uniform, but presents patches and gaps at various points in its course. The pigment lies of course under the retina, and is really choroidal; the pigment of the choroid being always denser in the neighbourhood of the disk. The disk itself is not exactly a transverse section of the optic trunk, but is rather the centre of dispersion, from which its fibres sweep in a cup-like expansion to form part of the retina, and the vessels which enter the eye at the disk sweep from it in the same way. Nor is it a raised spot, as the name 'papilla' would seem to signify, or indeed does signify; on the contrary, the healthy disk presents a trifling depression (*porus opticus*) about its centre, or rather at that point close to its centre where the retinal vessels issue. This part is extravascular and therefore whiter than the rest of the disk, and it consists of the connective tissue which invests the central blood-vessels. In slight cases of congestion this white centre, which generally appears to be of about the size of a small pin's head, shines out in contrast to the rest of the disk. In fuller states of congestion, however, this centre itself becomes flushed like the rest of the part, and I think it quite unnecessary, if not misleading or erroneous, to give a distinct name, 'peripapillary congestion,'

to the former appearance, as one recent writer at least has done.

I have said that the optic disk is of a rosy, or creamy white tint, and I shall now proceed to explain how this rosy tint, which varies according to the mode of examination, which differs a little in shade in various persons, and is often a little deeper towards the (real) inner half of the disk, is due to a special vascular system enjoyed by the disk in independence of that of the retina. If I am not mistaken, the disk and retina were supposed to draw their blood-supply from a common source, until Galezowski pointed out that the vascularity of the disk was independent of that of the retina, and rather formed a part of the vascular system of the brain. Since I became acquainted with Galezowski's views in 1866 I have gone carefully into this important question, and I have satisfied myself by means of many dissections, microscopic and other, of injected parts, and also by observation of the behaviour of the disk and retina in disease, that Galezowski in this important matter is mainly right. I say this important matter, for Galezowski's view, if well founded, is of great weight if it tends to establish still more a physiological division between the disk and retina, to explain more clearly their remarkable independence in disease, and above all to prove that the vascularity of the disk is a cerebral vascularity closely connected with that of the encephalon, and therefore a better guide to the state of the encephalon than the retinal circulation can be. The retinal veins and arteries, though scarcely brought into nutritive relations with the optic trunk, are nevertheless of course closely associated with it, for we found in tracing the course of the retinal veins and arteries that they penetrate the disk about its centre, and actually enter the optic nerve-trunk. The nervules and the vessels then pass through the sclerotic together, the vessels lying in the middle of the bundle, and so they pass on into the orbit, the artery indeed passing through the optic foramen, but the vein escaping a little sooner from the bundle and passing by the ophthalmic vein and sphenoidal fissure into the cavernous

sinus. Such a geographical relation between the optic nervules and the vessels is very curious, and unparalleled anywhere else in the body. This peculiarity, and the further peculiarity of the structure of the optic trunk, leads me to look upon it not so much as a nerve proper, but as a commissure or bundle of nervules. I look upon it, with its rich connective tissue and its rich and special blood-supply, as a more important offshoot of the encephalon than any ordinary nerve would be, and as likely therefore to offer more important indications of its varying states.

The optic nerves take their rise chiefly from the corpora quadrigemina, which bodies, and the anterior pair in particular, on grounds both of physiology and of pathology, we now regard as the principal, if not the only true centres of vision. Two little tracts or ribbons take their rise in the corpora quadrigemina and optic thalami, from a distinct nucleus, in which latter they receive a few fibres, cross the inferior surface of the crura cerebri, taking fibres from the geniculate bodies, and then detach themselves almost completely from the encephalic mass. After this detachment they become more cylindrical, they lie at the base of the brain, and the two tracts unite to form an elaborate commissure called the chiasma, from which again the 'optic nerves' branch off to each optic foramen. The remarkable decussations in the chiasma establish a direct commissure between the two eyes in front, and a crossed commissure between each eye and the visual centre of the opposite side, the remaining fibres being left to form a direct connection between each eye and the visual centre of its own side⁹. The chiasma is an oblong nervous mass of some size, seated in front of the sella turcica and the pituitary body. Its hinder horns run under the floor of the third ventricle. Behind it is the pineal gland. It will be seen at once that the nerves run along under that very part of the base of the brain where lymph is mainly generated in affections of the membranes of the base. The chiasma

⁹ I give the accepted view of the chiasma, which, however, is now called in question as regards the arrangement of some of its fibres.

has also very close relations with important blood-vessels. The coronary sinus, or a part of it, is situated close behind and below this body; the cavernous sinuses are in its immediate neighbourhood, and large cerebral arteries lie beside and in front of it. Now, according to M. Galezowski, the optic tracts and nerves receive several very important blood-vessels in their course. In the chief place, the optic tracts receive a very extensive investment from the pia mater, which covers and adheres to all its free surface; anteriorly, where the tractus is approaching the chiasma, nearly two-thirds of its circumference are clothed by pia mater, and farther back that membrane even insinuates itself a short distance between the posterior or inner margin of the tractus and the adjacent surface of the crus cerebri. The more special vessels of the central nervous organs of vision are described as follows by Galezowski, whose words are almost literally translated:—

‘1. An arterial branch entering at the posterior border of the testes, and distributing itself to them. This Galezowski calls the artery of the testes, or the posterior optic artery.

‘2. Four vessels of some size which pass into the optic tracts on the level of the posterior border of the cerebral peduncles, and so by a well-marked line divide the optic tracts from the corpora geniculata. Two of these vessels are arteries, and they may be called the vasa geniculata or middle optic arteries. They arise from the choroid plexus.

‘3. A rather large branch coming from the middle cerebral artery, and which passes to the corresponding optic tract. This may be called the anterior optic artery.

‘4. Finally, filiform branches passing from the pia mater to the chiasma.’

The veins are associated with the arteries, and need no special description¹⁰.

¹⁰ In addition to the vessels described by Galezowski, and which account for the well-known independence of retinal hyperæmia and discal hyperæmia, there is, on the other hand, at the nerve entrance a very curious connection

If in addition to these vessels we also bear in mind, as I have said, that the optic nerves and tracts are very extensively and closely invested by the pia mater, which gives off nutritive branches to them, we shall have a clear idea of the mode of this circulation which nourishes the optic tracts and nerves. Nor, when we look at the size and complexity of these parts, shall we feel any surprise at so elaborate a vascular supply. Now all these branches unite to form an uninterrupted network of vessels which extends from the tracts to the disk, and it is to them that the rosy tint of the disk is due. Its vascularity is mainly therefore a cerebral vascularity, and not an offset of the ophthalmic artery like the vascularity of the retina. I hope to show that these apparently small details bear closely upon the phenomena of morbid change as seen by the ophthalmoscope.

In accepting the description I have quoted above, I ought perhaps to add that it does not exactly coincide with nine dissections of the injected parts which I have myself made for the special purposes of this investigation. On the other hand, however, my own preparations did not quite agree among themselves, and in two cases I found a slight difference in the arterial distribution between one side of the brain and the other. At the same time it is not important now to discuss the matter as a mere question of minute anatomy, and if Galezowski's observations are taken in his own words, the reader will fall into no great error¹¹. Any

between the vessels of the nerve, of the retina, of the chorio-capillaris, and of the ciliary systems. It is not difficult to demonstrate by careful injections the existence of an arterial circlet without veins which surrounds the nerve in the sclerotic foramen. This circlet is formed by arterioles from the short posterior ciliaries; from it, again, minute twigs enter the nerve, and ramify in the canals of the neurilemma, where they anastomose with fine twigs from the central vessels. Fine branchlets also pass directly into the disk from the choroid, so that we get a kind of threefold continuity between the disks, the retina, and the chorio-capillaris, by means of an exquisite intraneural and perineural network. These vessels are not, however, of great nutritive value, or of much practical importance in this place; for further details, therefore, I refer the reader to the admirable researches into the vascular system of the human eye by Leber, '*Arch. f. Ophthalm.*' xi. 1, pp. 4-7 (1865).

¹¹ In a subsequent essay ('*Arch. Gen. de la Med.*' Dec. 1868) M. Galezowski accepts my views, published ('*Med. Times*,' May 9, 1868) almost in my pre-

variations which may hereafter be found to occur in the exact mode of the distribution in various brains, will not in any way affect the main question of a rich independent capillary circulation in the optic tracts and nerves extending up to the disks, and belonging to the cerebral system of vessels. In the optic disk, then, we have an expansion of the nerve fibres of the optic trunk, we have a capillary circulation continuous with that of the brain, we have the trunks of the larger and more independent retinal vessels, and we have a certain considerable quantity of connective tissue, part of which appears at the central depression and sheathes the retinal vessels; the remainder extends from the peripheral neurilemma inwards, and makes a framework for the optic bundle which I shall presently describe. As it passes backward from the disk to the orbit, the nervo-vascular trunk passes through a foramen in the sclerotic coat, which is at this point of entrance denser than at any other part of the eye. The opening seems to be guarded by a pierced plate, called the cribriform plate; this is really, however, an arrangement of the neurilemma, which here becomes continuous with the border of the opening, and exposes the open ends of the longitudinal canals. These sieve-like openings are to be seen through the transparent nervules in many healthy eyes, and in some cases of atrophy they become very evident. The margin of the foramen in the sclerotic forms an unyielding ring, called the sclerotic ring, and the nerve trunk as it traverses the sclerotic shows a slight constriction. It will be seen hereafter that the relation of this sclerotic ring to the optic trunk, with its nervules and vessels, is of great practical importance. As it leaves the eyeball and traverses the orbit, we find that the optic trunk is invested by a tough outer sheath, which is easily detached, and which seems to be continuous with the sclerotic on one hand, and with the dura mater on the

sent words two years ago, as corroborative in the main of his own. He states that M. Sappey has 'pleinement confirmé' his conclusions by his own researches, and quotes them in his lectures. Also that M. Fort embodies them in the last edition of his '*Anatomie Descriptive et Dissection*,' t. iii. (1868).

other. Whatever may be their genetic relationship, however, it has been shown by Sappey¹² that this sheath, though continuous with these structures, differs much from either of them. He shows that the optic sheath is rich in elastic tissue, which is not found in the dura mater or in the sclerotic, and that the nervi nervorum are peculiarly abundant in it. On removing the outer sheath of the optic bundle, we find below it a second sheath, also enclosing the bundle, but not to be stripped off from it. This inner sheath, which is continuous with the pia mater, is thinner and less compact in structure than the outer sheath, though more dense and fibrous than the pia mater.

The two sheaths, therefore, enclose a cavity which may be said to be continuous with the intermeningeal cavity of the brain, and which may perhaps become distended by the transference of any fluid effused at the base of the brain. This is confidently stated to be the case: I have had no opportunity of verifying the statement, and confess to some scepticism about it¹³. The visceral arachnoid, indeed, after accompanying the nerve fairly into the hole in the sphenoid bone, becomes reflected on the process of dura mater lining that aperture; so that the space between the sheaths cannot at any rate be continuous with the arachnoid cavity. The inner sheath not only encloses the optic bundle, but forms a part of it: it sends off processes or partitions which pass into its substance and divide it into

¹² Robin's '*Journal d'Anatomie*,' Jan. 1868. Vid. et Donders; Gräfe's '*Archiv.*' I. 2, p. 83.

¹³ This statement, which I doubted upon grounds of clinical experience, is now, however, proved by the accurate researches of Schwalbe (Schultze's '*Archiv.*' vol. vi. Part 1, 1870). He has investigated the eye by means of silver solutions, after Recklinghausen's method, and has demonstrated the existence of two lymphatic cavities, the one '*supra-vaginal*,' and extending around the choroid, between it and the sclerotic; the other '*sub-vaginal*,' occupying the space mentioned in the text, between the two sheaths of the optic nerve. It has, like the supra-vaginal cavity, the lining and other characters of a lymphatic cavity. Schwalbe shows that it is no doubt continuous with the similar interarachnoid lymphatic cavity, but towards the disk is closed, so that although it can be injected from the arachnoid cavity, yet the injection cannot enter the eye. Clinically, therefore, I was right, anatomically I was wrong.

numerous longitudinal channels, along which the filaments are carried. These partitions, then, are continuous with, or form the sheaths of, the fibrils themselves, a peculiar arrangement which shows again that the optic nerve is not strictly a nerve, but rather a bundle of nervules—‘a cylinder of collected tubes.’ I must lay some stress upon the disposition of this connective tissue, as it is greatly concerned in some of the morbid changes which I shall have to describe. If the reader is in any way familiar with the microscope, he will be able to demonstrate these details for himself. Let a healthy optic nerve-trunk in front of the chiasma be taken and soaked for about three weeks in a light straw-coloured solution of chromic acid. Thin sections of this are then easily made with a sharp knife, and rendered transparent by Clarke’s or Bastian’s methods¹⁴. These sections form beautiful objects, and their variations in disease are very interesting and easy to trace. The vessels lie in the connective tissue between the longitudinal canals.

From the foregoing descriptions it is clear then that the optic nerves are far more complex in structure and arrangement than any other nerves, and should rather be regarded as commissures or bundles of nervules; that they are highly vascular, and are vascular with a vascularity which is in close relation with that of the brain itself; that they are very rich also in connective tissue; that in their extensions into the encephalic cavity they traverse the base of the brain in the direction of parts which are continually liable to disease; that their course up to the geniculate and quadrigeminal bodies is a long one, and brings them into near relations with the important organs of the meso-cephalon; and that they have central connections in the geniculate and quadrigeminal bodies, which centres are again closely related to the cerebellum by the processus ad testes or superior peduncles, and to the great strands coming up from the spinal cord.

¹⁴ Oil of cloves is better than oil of turpentine in Clarke’s process; it is at least as efficient, and far more agreeable.

It is probable that the optic fibres have even more extensive connections than those which I have indicated—connections which have hitherto baffled the anatomist. The researches of Gratiolet, Schröder van der Kolk, and other most able investigators, have made it probable, for example, that some of the optic fibres radiate into the hemispheres, though they have found it impossible to decide upon the point. I believe, however, that the ultimate distribution of the optic fibres is less important to our present inquiry than is commonly supposed or appears likely.

ANOMALIES OF THE OPTIC DISK AND RETINA.

Before entering upon the chapter of actual disorder of the optic disk and retina, it is important that the observer should be on his guard against those insignificant, but often very striking, variations in the appearance of these parts, which depend upon mere individual peculiarities, and have no pathological meaning. Under this head of accidental anomalies, I shall consider the vessels first and the disk and retina in the second place.

I have said that the *distribution of the vessels* presents many differences, so that it may indeed be difficult to find two eyes exactly alike in this particular. Such variations, however, are not striking, nor are they likely to be mistaken for disease. A tortuosity, which is really a mere accidental peculiarity, is more likely to deceive, as it may lead to the inference of over distension of the vessels; or a twist or 'kink' in a vessel may simulate a hæmorrhage. This latter mistake is often made, and is as easily prevented by using a higher magnifier, or by looking at the erect image. The former is more difficult to guard against, and it is only after much experience that the observer should permit himself to express an opinion based only upon lesser changes in the colour, the size, or the tortuosity of retinal veins. A skilled observer, by considering all the points of a case, will know how to give their due weight, and no

more, to such appearances, whether evidences of disease or mere individual anomalies.

Persistence of the hyaloid artery must be a somewhat peculiar object, but it has never fallen to my lot to see it. In the fœtus an artery passes from the disk through the vitreous body to the lens, and in some rare cases this artery, or its sheath, instead of dwindling away, has been seen to traverse the adult eye. Laurence describes such a case in the July number of the 'Ophthalmic Review' for 1865, and many cases are recorded by German observers¹⁵. It is a mere curiosity, and needs no further description in this place.

Pulsation of the veins is not infrequently to be seen in a perfectly normal eye. It is a hydrostatic phenomenon, and is due to the impulse of the retinal arteries which compresses the vitreous humour, which in its turn compresses the veins, so that the venous pulse is synchronous with the arterial diastole and the cardiac systole¹⁶. The same effect may be produced artificially in any normal eye by slightly compressing the eyeball, and thus adding to the tension of the vitreous body. In some eyes the venous pulse is very evident without any extraneous pressure; but I believe arterial pulsation is never visible under any circumstances, natural or artificial. The venous pulse is generally to be seen in the large branches alone, in those which lie upon or lie close to the papilla, but in some cases I have seen it sweeping over all the field of observation. In the cases of physiological excavation of the disk which I am about to describe, the pulsation may be well seen in the bends of the veins as they climb over the edges of the cup. The stream is of course from the periphery towards

¹⁵ Vid. Liebreich, 'Klin. Monatsblätter' for same year, 1865, p. 24; Mooren, 'Ophthal. Beobacht,' 1867, p. 204 (Mooren's case was observed, however, in 1859); and Wecker, 'Annales d'Oculistique,' tom. 53, p. 65.

¹⁶ In the 'Arch. f. Ophth.' Bd. III. 2, p. 155, art. 'Ein Mikrometer am Augenspiegel,' Schneller demonstrates the variation of size in the choroidal vessels consequent upon changes of intra-ocular pressure. He shows, also, that atropine causes a diminution of intra-ocular pressure, and thus a distension of the choroidal vessels. Schneller's views seem, however, open to some question.

the centre, the dilatation moving in this direction and the contraction in the reverse.

The *excavation of the optic disk*, sometimes seen as an individual peculiarity, may well be mistaken for commencing disease, such as atrophy or glaucoma. In the normal eye, as I have said, there is always a slight depression near the centre of the disk, at the point where the vessels issue from it, and in some cases this depression may be so exaggerated as to become an actual cup. It is best seen of course with the binocular instrument. Here, again, we have to thank that successful and accomplished observer, Heinrich Müller, for the first adequate description of this peculiarity¹⁷. This excavation fortunately never proceeds far, though it frequently presents steep walls. It seldom sinks deeper than the thickness of the retina and choroid, and never trespasses upon the lamina cribrosa; nor, again, does it ever involve the whole disk, but preserves its original character as an exaggeration of the central pore¹⁸. The excavation is in some cases accompanied by other inequalities, a part of the disk being hollowed and another part elevated; or we may see a sloping wall on the side of the yellow spot, and a steep wall on the opposite side; or, again, the excavation instead of being circular may be irregular or angular. The changed colour of the disk in these cases is more striking than its change of form, and the strong redness of the disk around the cup contrasting with the white or the grey of the cup itself, may give a strong impression that disease is present. I have never seen the vessels bend under the edge of the cup so as to be lost to observation, as we see them in glaucomatous excavation, nor have I noticed any difference in the vessels themselves, except the slight difference in colour, which of course takes place in consequence of the changed reflections. These physiological excavations

¹⁷ Gräfe's 'Archiv.' iv. 2, s. 4.

¹⁸ I think it better in these and many similar passages to speak from my own experience, rather than to copy the statements of others. But I should say that other observers do describe this cupping as involving the whole disk, and as pressing beyond the sclerotic. So likewise with arterial pulsation, which some writers profess to have seen, and may have seen.

should be borne in mind, for I believe they are by no means uncommon. A friend of mine presents this anomaly in a marked degree, and I have had his case in mind while writing the above paragraphs. He narrowly escaped a terrible prognosis at my hands, for I discovered the state of his optic disk in the early and innocent stage of my inquiries.

No less important is it to recognise distinctly those *changes* which take place in *old age*, lest we attribute that to disease which is due only to lapse of time. In old age tissues lose their transparency, and undergo even further changes of a degenerative kind; the more delicate the part the more obvious, of course, such changes will be. As the skin loses its brightness and delicate vascularity, so the optic disk loses its transparency and the tints of its complexion. The dioptric media become dull, so that the back of the eye is less distinct, and the tracery of the disk and retina loses the sharpness and delicacy of its earlier and fresher life. Changes, therefore, which in youth would lead us to recognise an atrophic or even a sub-inflammatory process, in age would cease to have any such meaning. Practice alone can teach us to estimate these little variations at their true value.

Anomalies in the colouring of the disk. It is impossible to give any accurate idea of the colour of the disk by verbal description, nor have I made any serious attempt to do so. Almost every writer has given his own description of the tints of the disk, some more successfully, others less successfully; some evidently seeing with the general eye of mankind, others having peculiar visions of their own. But so many persons talk loosely about the disk seeming too white, the disk seeming too red, the disk having a bluish tint, or a grey tint, and the like, that I feel myself obliged here to show any person how to produce such anomalies of colour at will. First of all the colour of the disk differs a great deal in persons of various ages and of various complexions. I have just described the waning disk of age, and contrasted it with the fresh bloom of the more youthful nerve; I may add, that between persons of dark and light complexions

we may find at least as great a difference. This difference is due chiefly to the degree of pigmentation in the choroid, and the effect in the disk is mainly perhaps one of contrast. In deeply pigmented eyes the disk shines out with a luminous silver yellow light; while in eyes less rich in pigment the disk stands out less trenchantly, and usually seems of a reddish hue. The kind and degree of illumination again has a marked effect upon the tints of the disk. The strong yellow lights so often used, when combined with the bluish grey reflection of the nerve cylinders in the disk, may account for the greenish glimmer which some observers have curiously examined. This hard greenish appearance, which has aroused suspicions of atrophy in anxious minds, may be cured at once by adapting a pale blue chimney to the lamp. Again, the stronger the illumination the nearer in general the disk approaches to white. The delicate greyish blue so evident in the twilight of the direct examination, disappears in the glare of the indirect examination; under its strong light all delicate tints are banished, and the disk appears white or yellowish or reddish white. Again, in the direct examination the careful observer will even note changes in colour as he varies his accommodation. If we do not accommodate exactly for the surface of the disk, we shall see it uniformly of a bluish, yellowish or reddish white. But if we adjust our vision very accurately, and hold the mirror steadily, we shall detect a great difference between the inner and outer portions of the disk. Nothing is more common, however, than to see this difference pounced upon as an evidence of disease—‘the inner half of the disk was seen to be much congested,’ &c. In fact, the colour of the disk is compounded of the colours of its constituent parts—firstly, of the connective tissue, which is formed in the vascular sheath at or near the centre, and in the underlying lamina cribrosa; secondly, of the nervules deprived of their neurilemma; and thirdly, of the blood-vessels. The colour of portions of the disk varies, therefore, with the various arrangement of these elements. At the centre the connective tissue surrounding the vessels stands alone, and is and ought to be quite white. In the inner half of the disk

we have the other extreme. The nervules given off on this side are far more numerous than on the side of the yellow spots, and they therefore, together with their fine vascular networks, conceal the whiter element—the connective tissue—in the cribriform plate. On the other side, near the yellow spot, there is a much thinner layer of expanded nervules, and more light is therefore reflected from the white cribriform plate. So distinct is the cribriform plate on this side, that in the erect image spots and streaks can be seen which correspond to the canals from which the fibres issue. If we look perpendicularly into the canals, they appear to us as greyish dots; if we look obliquely into them, they appear rather as oval marks or streaks. In wasting conditions these stipplings or streaks become much more evident, and may be seen in the inverted image. Some rare cases are recorded in which the disk was of a strange colour, blue, for example, or red brown, and in which vision was unaffected. When pigment invades the disk, it is always black, and is probably accidental. It has rarely happened to me to see it upon the disk itself; it is generally confined to its outskirts.

There is yet one more striking kind of abnormality in the fundus of the eye which has often caused much perplexity to inexperienced observers, and does certainly suggest an alarming state of disease. I refer to the *white patches* which are sometimes seen upon the edge of the disk and invading the retina. They are not, I think, very uncommon; they may occur in one eye or in both, and they may be single, or two or more may be present. They are based upon a segment of the margin of the disk, and may extend upwards, downwards, or inwards; they seldom extend towards the yellow spot, for reasons which will be evident when we have explained their anatomy. They shine with a pearly white or greenish lustre, and have, as v. Recklinghausen¹⁹ says, an asbestos-like appearance, especially in those cases in which they present a striated character. This striated character may indeed always be made out by the direct examination, and is an important point in

¹⁹ v. Recklinghausen, 'Markige Hypertrophie der Nervenfasern der Netzhaut.' Virchow's 'Archiv.' Bd. xxx. s. 375.

their diagnosis. Their borders are jagged or irregular, and generally shade off a little into the neighbouring retina, which by contrast appears very red²⁰. They do not interfere with vision; they are always congenital, and in some animals are a part of the normal state. In these cases streaks may sometimes be seen along the borders of the vessels; and such streaks, taken together with the patch or patches, might lead an unwary observer to suppose the presence of albuminuric retinitis, though the appearance is really very different. The vessels which traverse the white patch stand out, of course, with great distinctness when they pass over its surface; when, however, they pass along in the thickness of the patch, they are more or less concealed; or they may pass along at various depths in its substance, when they appear thicker or thinner according to the depth at which they are embedded. This strange abnormality depends upon an extension of the sheath of the nerves beyond the cribriform plate, the patch being, in fact, made up of nontransparent insulated nerves. The normal transparency of the retina, as we know, depends upon the arrest of the sheaths of the nerves at the optic disk; and if the sheaths be accidentally continued beyond this point, we have an opaque white patch corresponding to the number and length of the fibres so sheathed. The neurilemma thins off at the borders of the patch, leaving the axis to pass in the usual way.

We shall now proceed to the consideration of those changes in the optic disk and retina which do signify the presence of disorder; but I shall confine myself to the changes which have a symptomatic value. For a description of the changes which, like glaucoma, are evidences of local disease only, I must refer my readers to the works of ophthalmic surgeons.

²⁰ Vide Liebreich, 'Atlas,' p. 27. Plate xii. figs. 1, 2.

CHAPTER IV.

ON THE VARIATIONS FROM HEALTH OF THE OPTIC NERVE AND RETINA.

IN this chapter I propose to describe the morbid changes which take place in the optic nerve and retina, in order that, when they are themselves fully understood, their connections with other disorders may be more clearly discussed hereafter. According to the canon upon which I often insist, we shall best interpret these morbid states by tracing their origin in the slightest deviations from health. The first class of changes I shall describe are those which occur in the circulation, and which are not attended with any sensible deterioration of structure; these we shall not refer to a standard of 'optic neuritis,' but to the standard of health; and we shall try to detect the early and various modes of departure from that state. After discussing the simple variations in blood supply, we shall pass on to congestions with effusion, to neuritis and atrophy.

The parts which we have to watch are the optic disk, the retina, the choroid, and the blood-vessels. The optic disk is liable to anæmia, to simple congestion, and to congestion with effusion within or around it; to inflammation of its outer sheath, to inflammation in its substance or inner sheath, and lastly to atrophy. The retina is liable to serous, fibrinous, and fatty exudations or patches, more especially in the course of the vessels; also to hæmorrhages. The choroid

is liable to loss or disturbance of its pigment; also to hæmorrhages. The blood-vessels are liable to many characteristic changes—to diminutions or obliterations, to dilatations, to tortuosities, to pulsations, to varicosities, to blood stases, embolism, and thromboses, to diseases of their coats, and to rupture. The reader will scarcely expect me to enter into minute descriptions of these very various states. I shall only describe the meaning and causation of the chief deviations from the normal condition. The great variation which is found even in healthy nerves is one of the difficulties experienced by beginners in ophthalmoscopy. Let me earnestly repeat, that many peculiarities which to the unwary observer appear to be marks of disease, are in no way of evil meaning. Not only, as I have said, do we find from time to time such peculiarities as large white patches upon the retina, and white rings or rings of pigment upon the margin of the disk, which varieties may be congenital or may be mere harmless changes, having no special meaning; but we find variations also in the colour and vascularity of the optic nerve, which at times may be puzzling even to a practised observer. There is sometimes room for doubt whether a deeply coloured disk is due only to the youth or the complexion of the patient, or whether it be due to congestion. A pale disk, again, may be pale from general or local anæmia, or its pallor may be the mark of commencing atrophic change; in one person some largeness of the vessels and distinctness of the capillaries is a physiological condition, in another it may be due to pathological conditions. Nothing is more blameable than the off-hand condemnation of optic disks as 'too red,' 'too white,' and so on. To determine the presence of slight congestions in the papillæ is not easy, and can be done only by carefully considering all the circumstances of the case, by watching its progress, and by comparing one eye with the other.

In the normal state the eyes are generally alike, but in morbid states it is rare for the two eyes to advance by quite equal degrees, though a few instances are recorded in which one optic disk has been destroyed by atrophy while the other remained healthy. I have seen this in three cases. In cases

of encephalic disease the disk on the same side as the disease is sometimes affected first and chiefly.

A. *Hyperæmia.*

The main distribution of the artery and vein of the retina are tolerably uniform, and as they are very liable to change in disease it is of great importance that the observer should accustom his eye to their average sizes, tints, directions, and frequency at various ages. The colour of the disks should also be carefully watched in a large number of healthy persons, so that the eye may not discover signs of morbid change in normal peculiarities of age, complexion, and other modifying conditions. Besides the peculiar vessels of the disk and the peculiar vessels of the retina, there are certain other vessels which I have been long accustomed to watch and to describe as the radiating vessels of the disk. They are in nutritive relations with the disk and also with the retina and choroid; and in atrophy of the disk they may be seen to shrivel, grow tortuous, and vanish, while the retinal artery and veins are almost unaffected. In states of congestion, on the other hand, they appear in numbers, and radiate in a star-like manner from the disk, chiefly on its lateral aspects. They have few branches or offsets, and in healthy conditions, under ordinary magnifying powers, they are only visible in twos and threes. Their appearance, therefore, in any number is significant of slight degrees of congestion.

In the higher and quite unmistakeable degrees of hyperæmia, on the other hand, we are in danger of confusing the secondary neuritic processes, which may complicate it, with descending neuritis. This distinction is, however, at least as important as the distinction of early hyperæmic stages, both as regards the presence of a certain symptom and its meaning, and also as regards its danger to vision. In secondary neuritis of the papilla following congestion, the prognosis as to vision is far more favourable than in 'descending neuritis,' and in acute descending neuritis more favourable than in chronic neuritis or progressive atrophy. Simple hyperæmia

may occur in the papilla, in the retinal veins, or in both retina and papilla together, which shows the independence of the two circulations. At first the leading vessels, and chiefly the retinal veins, grow fuller, darken in colour, and tend to become a little tortuous, or even varicose. They seldom give way except in cases of albuminuria; and in these latter cases the blood effused degenerates more or less quickly, so that the hæmorrhages appear as brownish or whitish blotches or streaks across or along the course of the vessels. These marks are very characteristic of that state of the system in which the small rough kidney is also formed. In retinal congestion the extremest vascular branches may also be followed with greater ease on account of their dilatation and darker colour; for the same reason they seem also more numerous, and the distinction between venous and arterial branches becomes more difficult. This point is an important one, for in the hyperæmia which occurs in consequence of disordered accommodation, we have no difficulty in detecting the arteries, but often rather a greater ease.

To pronounce upon the lesser degrees of hyperæmia must always be a most delicate and difficult task; but it is a great help to us if, as is most usual, we find these changes more advanced in one eye than in the other; and Galezowski asserts that the eye on the side of the brain mischief is commonly the worse—an observation which I too have made in some cases, but which is not, I think, universally true. Pulsations, which may sometimes be seen in the veins of the normal eye, especially if a little pressure be made upon the ball, are more evident in hyperæmic states. Examination by the direct image is important in these cases. Congestion of the disks themselves is generally first seen on the inner half, where the small vessels are more numerous, and from thence a full red invades the whole papilla. Slight œdema may then take place, so that its edges are dimmed, and the disk is veiled by a cloud, or its outline becomes altogether lost, and its locality known only by the convergence of the veins. In earlier stages, however, the connective tissue about the vessels in the centre remains white and strongly contrasted with the

red periphery. Sometimes little dark red spots are seen upon the angry-looking disk, and are called ecchymoses; when examined, however, in a larger image, they may nearly always be resolved into little dilatations or kinks in the vessels. This kind of disk is quite independent of any hyperæmia of the choroid, and indeed may sometimes occur without much dilatation of the retinal vessels. The sight may be dimmed, especially if there be a film of œdema, the eyes may feel heavy, or the patient may complain of flashes of light, iridic colours, and the like. Neither these conditions, however, nor neuritis cause photophobia, which seems to be due to the pain of movements in the ciliary region and to depend upon the fifth nerve. We need not fear, therefore, in spite of Jäger's warning, to use a strong light, or to make repeated examinations in this state of the optic disks.

The *causes of hyperæmia* are many. It may be the first stage of full ischæmia, of neuritis, or of an atrophic process; or, again, it may be due to orbital disease, to choroiditis, or to Bright's degeneration. Slight degrees of it are not uncommon in drunkards; but in a very great number of cases it is due to encephalic disease—to tumour, to acute or chronic meningitis, or to changes in cerebral vascularity, which may be attended with convulsions or mania. The presence of hyperæmic and anæmic conditions of the disk and retina in convulsive and maniacal diseases is of high pathological interest. In epileptics I am satisfied that there is a higher average fulness in the vessels of both disk and retina than in healthy persons. It is difficult to assert this in any single case; but if we take a hundred cases of well-marked simple epilepsy together, we shall see a higher contrast in the white centre of the disk, a deeper and perhaps stippled redness of its circumference, a purpleness and distinctness of the veins, and a frequency and decision about their smaller branches. They are seldom tortuous; nor are the outlines of the disk often obscured, though this was the case in Ann G——, No. 5, Appendix.

Transient hyperæmia may be seen in heart diseases which obstruct the venous circulation, and in Graves' disease; but I

do not know how far we may reason from it to the encephalic condition. In Graves' disease, indeed, the venous turgescence seems to me more likely to be due to pressure upon the internal jugular vein. Venous hyperæmia of the retina may be seen, too, in menstrual disorders. Inquiry must always be made, therefore, into the state of the heart and other functions before we decide on the symptomatic value of intra-ocular congestions.

It is very frequently stated, and stated with a confidence which is quite unjustified by any complete or accurate observation, that the sympathetic nerve system is endowed with great power over the vascularity and nutrition of the optic disk and retina. Fancies about the sympathetic nerves are now very fashionable, and their mysterious agency is called upon every week, every month, and every quarter to explain all sorts of phenomena, or supposed phenomena, in healthy physiology, morbid physiology, and therapeutics. The less the writer and the reader know about the sympathetic nerve system, the more satisfactory, of course, is the explanation. It is asserted in most ophthalmic treatises that paralysis of the sympathetic in the neck causes hyperæmia of the optic disk and retina. This may be so: indeed, I am far from saying that it is improbable; but, so far as I know, it is wholly unproven; nor do I find any proof of this assertion in the writings of those who repeat it. Bernard certainly says nothing of the kind; and in cases where I have been able to examine the back of the eye during conditions of disorder in the cervical sympathetic, I have found no consequent hyperæmia. I have examined the eye carefully with the ophthalmoscope in two cases of aneurismal pressure upon the cervical sympathetic, in three cases in which the cervical sympathetic was paralysed by disease of the neck, in many cases of Graves' disease, in which latter disease the sympathetic is said to be at fault, and in many cases submitted to galvanism of the sympathetic in the neck; but I have obtained no constant results. There is sometimes *venous* hyperæmia in Graves' disease, but this I should refer, as I have said, to venous obstruction rather than to the paralysis

of the carotid. Few conclusions command my wonder more than the common accusation of the sympathetic nerves in the so-called 'spinal amaurosis.' If the sympathetic can set up an atrophic process, no doubt it may easily establish hyperæmia; but I have yet to learn that there is a single valuable clinical fact to be adduced in favour of either process. Should it turn out that section of the ciliary nerves prevents the occurrence of sympathetic ophthalmitis, we shall no doubt have done something to enlighten a very difficult subject; but, so far as I am able to tell, the conjunctiva and cornea seem to be the first to suffer in the implicated eyeball, as they are the first to suffer in sections of the fifth nerve. When I treat of amaurosis in spinal disease, I shall have to return to this subject.

B. Anæmia of the Disk and Retina

is the opposite of hyperæmia, and depends upon an emptiness of the vessels. I am sorry to see many authors whose words have weight using the word 'anæmia' when they mean, or may mean, atrophy of the disk. It is of great importance, both for the physician and for the ophthalmic surgeon, to distinguish the two states, nor do I think it is often difficult to make the distinction. Anæmia of the disk is nearly always accompanied with anæmia of the retina and choroid, so that anæmic eyes light up badly; while in atrophy of the disk the choroid may be of healthy brightness. The retinal vessels, too, in anæmia, are shrunk—shrunk to a degree we should not find in commencing atrophy¹, and an anæmic disk never has the hard, sharp, staring look of atrophy of the third degree. In atrophy of the first and second degrees, if subsequent to neuritic changes, the retinal vessels would be rather swollen than collapsed; and in anæmia we seldom or never see the vessels standing out so distinctly against the paling background of the disk as we see in atrophy. In anæmia, too, it

¹ There are some good drawings of the anæmic fundus in the Library at Moorfields. In one case the anæmia was connected with irregular menstruation, in the other with oversuckling.

is generally possible to distinguish the arteries from the veins, which distinction, in atrophy, is more commonly lost. Again, the edges of the disk in anæmia are not so sharp as in early simple atrophy, the fibrous extensions to the retina remain uninjured, and under a good light, and with four or five-inch lens, a fibrous texture may, in anæmia, still be detected; the more as I think there is often a slight œdema in anæmic disks making this coarseness of texture more evident from the swelling of the nerve filaments. The œdema also gives a greyish look to anæmic disks. These phenomena, however, are studied best in the erect image².

In atrophy the nerve fibres waste, and are replaced by connective elements. Atrophy, again, is generally unequal on the two sides, while anæmia is equal; and atrophy does not, as a rule, begin all over the disk at once, but invades the disk, as the arcus senilis the cornea, or works across from the outer to the inner moiety. The subjective symptoms, too, are generally different. In simple atrophy we are told of a gradually increasing amblyopia, attended with scotomata; in anæmia, of capricious fits of darkness—of sudden blindness on rising from bed, for instance—relieved by intervals of fair sight, and instead of scotomata we hear of flashes of light, sometimes of a most painful intensity, or of ‘*musæ volitantes*.’ The field of vision, again, in atrophy nearly always contracts from the internal side; in anæmia there is uniform feebleness of vision all over the field. These considerations, taken together with the history of the case and its general symptoms, will always, I believe, help us to a pretty certain conclusion.

The *causes of anæmia of the disk and retina* are the same as those of general or local anæmia, or the anæmia may be due to vascular spasm. Cases 6, 12, 14, 18 in the Appendix are good examples of anæmia. We must be careful to remember that transient blindness or dimness may be due as well to transient anæmia of the perceptive centres as to anæmia in

² I must warn the inexperienced observer that the retinal vessels, when seen against a whiter disk, appear larger by contrast. Allowance must be made for this, and, after some practice, such allowances are made almost unconsciously.

the eye. The dimness of vision which often occurs during aortic regurgitation may be due to either or to both states. I have often seen anæmia of the eye in these cases. In some cases, again, we seem to have an epilepsy of the function of the retina due to anæmia, as we have epilepsy of the function of the corpus striatum in animals bleeding to death, and as we have an epilepsy of the mental function in mania. I have now a man under my care who is subject to epilepsy, and who has 'fearful flashings of fire' in his eyes by way of warning. Dr. Hughlings Jackson also has published such cases, which, in fact, have been noticed from the time of Aretæus. The occurrence of vascular spasm and paresis in a visible part like the eye is, if finally ascertained, a fact of very high interest, as it would raise our notions of a like morbid process in the brain from a probable hypothesis to a very safe inference. In its effects upon function, venous hyperæmia is very similar to anæmia. I have given in the Appendix a case of convulsion during which I found a hyperæmic state. [Case No. 5.] I found the reverse condition in another case in which I watched the disks and retinas during a long-continued status epilepticus. In this there was marked anæmia of the disks, trespassing a little upon the neighbouring fibrous coat of the retina, and Mr. Carter tells me that in such a case he once noted the same appearance.

I believe that there is an important distinction between partial and complete anæmia in these cases. Complete anæmia, as in embolism, will probably abolish function, while partial anæmia, due to vascular spasm, to bleeding, to pressure, &c., allows of the accumulation of force at a low tension, which is irregularly discharged as energy. So it is also with partial and complete degrees of venosity of the nutritive blood; but to this interesting question I must return at another time.

C. *Œdema of the Disk.*

Of simple œdema of the disk and neighbourhood I have seen little or nothing, but the subject is one of some interest. Œdema very often occurs as a complement of other affections,

as, for instance, of neuritis, of anæmia, or of embolism of the central artery; in neuritis, indeed, it plays a considerable part, and is the cause of that 'woolliness' which Mr. Hutchinson often describes. It has never happened to me, however, to see œdema as a substantive disorder. Manz³ speaks of the occurrence of enormous dropsy of the nerve-sheath in some cases of optic neuritis, or rather of ischæmia papillæ (vide Case No. 34, App.), but does not speak of it as existing alone. The most interesting observations upon this subject which I have met with are by Macnamara, who relates in detail a case in which œdema of the disks was a prominent and most important symptom⁴. The patient was a little girl aged thirteen, who had suffered from quotidian ague. As the ague disappeared under treatment, she became palsied of all four limbs, of three limbs completely and of the left arm partially. Reflex action remained, and there were no abnormal sensations. There was no disease elsewhere, except an enlarged spleen. So far, the case might well have been one of so-called hysterical paralysis; but the state of the disks contradicted such a diagnosis. With the access of the palsy there appeared also some dimness of vision, which rapidly advanced to blindness, with dilatation of the pupils. With the ophthalmoscope there was found no neuritis, no retinitis, no atrophy, no hæmorrhage, no interference with the arteries or veins, but there was great œdema of the disks. Macnamara assumed that there was a similar state of serous effusion without structural mischief in the centres of motion; and the results justified his assumption. Under the use of iodide of potassium with strychnine and arsenic, the girl made a perfect recovery in a short time, her sight and motion being quite restored. Macnamara has noticed these conditions more than once, and believes that malarious disorder is a disposing cause. A like state of things seems to occur in the horses and cattle of Western Europe. A very intelligent and skilful veterinary surgeon, Mr. Fearnly,

³ 'Klinische Monatsblätter' (1865), p. 283; quoted by Mauthner, 'Lehrbuch,' p. 290.

⁴ 'Medical Times and Gazette,' May 2, 1868.

of Leeds, tells me that by reading my papers on the use of the ophthalmoscope, he was led to use the instrument in the nervous diseases of animals, and has obtained some valuable results. In particular, he tells me that in several cases he has met with simple œdema of the disk and neighbourhood, coinciding with curable palsies of the limbs. Two cases he related to me which much resembled Macnamara's case, and in them also he obtained a speedy recovery by the free use of iodide of potassium. Mr. Fearnly had not met with Macnamara's remarks, his own observations being quite original.

D. *Ischæmia of the Disks.* (Choked disks⁵.)

Before I had long used the ophthalmoscope in cerebral disease, I began to entertain serious doubts about the true neuritic origin of many extreme disturbances of the disks. I gradually became assured that many of the worst cases of so-called optic neuritis are really mechanical congestions or venous arrests, differing essentially and importantly from inflammations. I attributed this stoppage to pressure upon the cavernous sinus, and supposed that such pressure would account for all the phenomena. I afterwards read the well-known paper by Gräfe ('Arch. Ophthalm.' 1866, ss. 114-119), where this great oculist proves that congestion and swelling of the disks with effusion are often due to vascular arrest alone. He points out, however, that this could not result simply from obstruction in the cavernous sinus, but must depend upon the concurrent action of the sclerotic ring. We have just seen that this unyielding ring so accurately fits the nervo-vascular trunk which traverses it, that when the slightest venous arrest distends this trunk, its embrace becomes a strangulation. He shows, accordingly, that these congestive affections of the disk are in the first place, or are throughout, confined to that part. ['Beschränken sich die Veränderungen (starke Schwellung und venöse Stauung) nur auf das intrabulbare Sehnervende.'] Many of our de-

⁵ Vide Liebreich, 'Atlas,' Tab. xi. fig. 2, and the frontispiece of this volume.

scriptions of 'optic neuritis' have been taken from this state of strangulation, which I would propose to call 'ischæmia papillæ.' This action of the sclerotic ring enables us to form most accurate opinions upon degrees of pressure within the skull, as, to use Gräfe's happy expression, 'it plays the part of a multiplier' placed upon a vascular offshoot of the brain. Ischæmia of the disks may often, but cannot always, be distinguished with the mirror from optic neuritis, as the two are frequently associated. I shall compare the two conditions when we come to 'optic neuritis,' and shall now try to describe simple ischæmia papillæ in the third degree. The trunk of the nerve is unchanged, and all the morbid signs are confined to its intra-ocular termination. This part, we see, is greatly swollen, and it generally rises steeply on one side, and sinks gradually to the level on the other. A skilful observer will be able to detect this projection of the disk, both in ischæmia and neuritis, by the shortening of the axis of the eye in this direction. Such projecting disks may often be seen, as in hypermetropia, by the mirror alone. There is some swelling also of the fibres themselves, so that they lose their transparency, and the papillary region looks more coarsely fibrous than in health. Its colour is often a mixture of dirty grey and red, due to the mingling of passive effusions with distended capillaries and hæmorrhages, but in other cases there is not much extravasation of blood, and the protruded disk looks bright or almost transparent.

Small patches of extravasation from rupture are, however, commonly found in numbers upon the disk. The morbid appearances trespass a little, but not far, upon the retina, seldom to a distance of more than half the diameter of the disk. The margin of the disk is wholly concealed by infiltration; by excessive vascularity, which gives it a mossy appearance; and by the coarsened fibrous extensions to the retina, which in the erect image give a striated quality to the disk and the peripapillary halo. The opacity of the retina rapidly ceases from this point, and there are no films or degenerative patches beyond, except perhaps

streaky exudations in the course of some of the larger veins. The veins of the retina are enlarged, sometimes enormously, and they tend to become very tortuous both in the plane of observation and from before backwards; they may also be very varicose. I have never seen them ruptured in ischæmia, nor are they so much concealed by exudation as they are in neuritis. I have twice examined disks in this state after death with the microscope, and have found them to be as described by a few other observers. The disks are enlarged and thickened, and the swelling and thickening extend more or less into the fibrous and other layers of the retina. There is exudation into the substance of the disk, and its vessels are enlarged, distorted, and in many places thickened. There is some cell and nuclear proliferation in the course of the vessels, and in the cribriform plate. In the fibrous layer of the retina may be found the homogeneous bodies without limiting membrane or nucleus, which probably result from the breaking up of nerve tissue⁶. A very interesting examination was made by M. Cornil upon a case under the care of M. Vigla. The case was one of encephalic tumour, in which the worse symptoms had rapidly developed themselves, and the disks had not, in all likelihood, been long congested. M. Cornil found only a prominence of the disks, serous infiltration of the connective tissue, and a few small hæmorrhages⁷.

The extra-ocular parts of the optic nerve are normal. What we find, then, is some 'inflammation' of the disk and retina immediately around it, as shown by proliferation of cells from the neurilemma and the sheaths of the vessels, and development of new vessels, with disintegration of nerve fibres. Nor is this contrary to expectation. I do not hold with Gräfe that this inflammation results from the greater susceptibility of the congested structure to ordinary 'irritations,' nor that the extravasations of blood are 'foreign

⁶ Virchow's examinations in Gräfe's cases are to be found in the paper to which I have referred. See also Sämisch, 'Beiträge zur normalen u. path. Anat. des Auges.' Leipzig, 1862.

⁷ 'Arch. Gen. Med.' (Dec. 1868), p. 679.

bodies' and sources of local 'irritation.' I think it better to say that the 'inflammatory products' are due, first, to the great disturbance or arrest of nutritive relations, and, secondly, to mechanical lesion followed by greater or less resistance. Sir W. Jenner very well describes the sub-inflammatory results of congestion of tissues in his paper upon 'Congestion of the Heart⁸.' The irritation at the disk is sometimes propagated beyond the lamina cribrosa up the orbital portion of the nerve, making a '*neuritis ascendens*.'

It is astonishing how changed and disfigured the optic disk and neighbourhood may become in this affection without disturbing central vision. I have lately had several such patients under my care who could read a badly printed news-sheet with ease. The same fact is strongly insisted upon by Gräfe. For this reason the condition is constantly, I may perhaps say generally, overlooked, unless it should happen to come before an ophthalmic surgeon. The prognosis as to sight is also better than in descending neuritis, but both affections point too surely to serious encephalic mischief. The microscope shows, however, that in ischæmia many more nerve fibres retain their continuity than is the case in neuritis⁹.

The causes of ischæmia papillaris are all those changes within the skull (I shall omit all discussion of orbital causes¹⁰)

⁸ 'Med. Chir. Trans.' vol. xliii.

⁹ The causation of ischæmia has been much discussed in the German periodicals since the above was written, especially by Hermann, Schmidt, and Schwalbe. See, particularly, an article by Schmidt in the 'Arch. f. Oph.' vol. xv. Part 2, pp. 193-197. Schmidt denies Schwalbe's statement (vide p. 35) that injections of the arachnoid space fill the capsule of Tenon; and still less, he says, do they fill any perichoroidal space. He asserts, however, that they do fill the intravaginal space of the optic nerve, and that the injection there empties itself into a 'canal system' which ramifies in the lamina cribrosa. He concludes therefrom that the *Stauungs papilla* arises from increased intra-ocular pressure, due to *Stauung* of the injected fluid in the canal system continuous with the arachnoid cavity. These observations need verification, but if proved are singularly interesting, as showing the connection between affections of the base of the brain and congestions of the disk.

¹⁰ Mr. Salter, in the 'Med. Chir. Trans.' and 'Guy's Reports,' has given some most interesting cases of amaurosis following inflammations arising in

which more or less directly distend the ophthalmic veins. Distensions which, in other veins or in other branches of the ophthalmic vein, would be scarcely noticeable, are, by means of the multiplying action of the sclerotic ring, made very manifest in the branches of the retinal vein, and present the appearances I have described. The three main causes of ischæmia, with the subsequent atrophy, are: (1) meningitis; (2) hydrocephalus; (3) tumours. It seldom or never results from acute or chronic softening of the cerebral substance, from hæmorrhage, from sclerosis, or from arterial degenerations. It probably occurs in caries of the base of the skull, though I have not seen it: I have always seen neuritis in these cases. As, however, the causes of ischæmia may also be causes of optic neuritis, I shall now proceed to describe optic neuritis.

E. *Neuro-retinitis*¹¹.

I will now ask—first, what ‘optic neuritis’ means as a name; and, secondly, whether that meaning includes all the kinds of change attended with increase of vascularity to which the optic disk is liable. Now, if our medical terminology be worth anything, ‘optic neuritis’ should mean, or rather should answer to, inflammation of the optic trunk. What, then, is inflammation of the optic trunk? or, in the manner of Pilate, what is inflammation? Surely the conception of the movement known as inflammation, or which alone ought to be known by that rather objectionable name, is sufficiently simple. In lectures, I am wont to define ‘inflammation’ as *lesion with reaction or resistance*. This excludes all transient disturbances of molecular tension, without disintegration. The idea of inflammation must start from a rupture of continuity, however

the jaw. He asks how the atrophy is caused, and I hope that the foregoing explanation of the strangulating power of the sclerotic ring may make this clearer.

¹¹ Vide Liebreich, ‘Atlas,’ Tab. xi. fig. 11; cf. also Tab. viii. fig. 6.

minute, and we must regard the subsequent congestion and other phenomena of resistance as secondary. Resistance will manifest itself in various ways, according to the conditions of the ruptured tissue. When this is surrounded by vascular and nervous connections, we shall observe not only cellular resistance, but also nervous and vascular resistance. We shall see proliferation, congestion, and heat in their various degrees, according to the complexity of the part which suffers. Vascular extensions do not make inflammation any more than the railways of the force in Abyssinia made the war; they are merely the evidence of lesion, and the conditions of resistance to it in tissues of a given complexity. And yet all severe congestions of the optic disk, with their consequent effusions, are called optic neuritis! The truth is, we cannot shake off our ontological conceptions of a 'nature,' an entity, I believe, of the female gender, who is always planning something in the human body—'eliminating morbid poisons,' plugging up inappropriate perforations, 'setting up inflammatory actions,' and so on. It is really time we avoided all this reasoning from final causes, and that we sincerely regarded the functions of tissues as the evidence of an equilibrium mobile which possesses greater or less powers of resistance according to its tension, and which manifests such resistance variously according to its complexity.

If we pass a ligature round the ophthalmic vein, we produce thereby great congestion of the optic nerve behind the ligature, and an escape of the vascular contents, due not to increased attraction on the part of the tissues, but to mechanical causes, causes like those which, in states of portal obstruction, favour an escape of serum into the peritoneal cavity. I have described this change as *ischæmia papillæ*, and it is not only incorrect, but very misleading, to call this result 'optic neuritis.' Injury to tissue is not the starting-point of the disturbance, though, of course, a secondary neuritic process with increased proliferation of the connective elements may be set up in consequence of the pressure. This is no idle or merely verbal distinction. I have pointed

out in the section upon papillary ischæmia that severe discal congestions, not neuritic in nature, are common—commoner perhaps than true neuritis—and as the nerve tubes may be little injured, it may cause but little alteration of vision. Such states, therefore, are seldom presented to the ophthalmic surgeon, and are to be discovered rather by the physician, whose mind moves in the opposite direction. In true ‘optic neuritis,’ on the other hand, the connective elements suffer first, and the subsequent congestion is slighter in degree, though the vision, for obvious reasons, may fail sooner. True optic neuritis, however, may go far without fully arousing the patient’s attention.

One main distinction between ischæmia and neuritis optici is, that while the former affection is, as I have said, confined to the disk, the latter affects the nervous trunk in a greater or less part of its length. It is, therefore, often called *descending neuritis*. This process is one of very great interest to students of nervous diseases. In it we see the mode of inflammatory destruction of nervous tissue, and from it we may draw some valuable inferences as to the mode, the rate, and the propagation of like changes within the cerebro-spinal cavity or in the course of other nerves. The reader is no doubt aware, for example, that secondary neuritic changes have been found in the nerves supplying the limbs in some of those cases where paralysis has been followed by contraction. In descending neuritis the connective tissue of the nerve is probably the active agent, the nervous elements suffering by implication. In the eye the vascular changes are secondary, and in uncomplicated neuritis there is no pressure upon the cavernous sinus. In meningitis, however, neuritis optici is often complicated with, or preceded by, ischæmia, as the inflammatory change may invade both the nerve and the membranes which form the sinus, so that it becomes choked with coagula or by the accumulation of exudative products above it, and a differential diagnosis becomes impossible. Pure neuritis presents the following appearance—the nerve is swollen, but less so than in ischæmia, and it does not present that steep elevation of one side so characteristic of

ischæmia¹². The vessels, again, are of somewhat different appearance. There is not the same bursting into view of a multitude of minute branches and capillaries which may give so mossy a look to ischæmia. The distension in neuritis is more an enlargement and tortuosity of the main trunks, though of course there are many more vessels to be seen than in health. As in ischæmia, the arteries become thin and indistinct, and there may be numerous minute hæmorrhages in and near the disk. The colour of the parts, again, is distinctive in well-contrasted cases. In neuritis we do not see a circumscribed intense redness or brownish-grey, but rather a wash of reddish-lilac, or a grey tint, and the tint, which is more uniform and more opaque, also extends more widely upon the retina than in ischæmia, and conceals more or less even of the large veins which converge towards the disk. The vessels, especially the veins which lie deeper embedded in the dense new formation, dip in and out or dive wholly out of sight for more or less of their course. The parts often have, too, what Mr. Hutchinson calls a 'woolly' appearance, due perhaps to œdema. Gräfe considers that this neuritis is not confined to the fibrous layer of the retina, but that all its coats are affected. He rests this belief upon the persistence of white patches, the implication of the yellow spot where the fibrous coat is not, and on microscopical researches. I have certainly seen in two cases a general retinitis depending upon cerebral disease. In some cases of neuro-retinitis the hæmorrhages bear a great proportion to the other changes, so that the disk and neighbourhood have a very bespattered appearance in the earlier stages, and in the later ones there are numerous white spots and other marks of much strife about the stricken disk. I believe this form is not a separate process, but depends in some cases, perhaps, upon the co-existence of extreme intracranial pressure, as in tumours of the middle fossa, and in others upon

¹² These swellings, making the disk actually a papilla, are best seen with a binocular instrument. A skilled observer will estimate them in the erect image by slightly varying his concave lenses, and after some practice we learn to infer their existence in the inverted image by the disturbed reflections.

the ready yielding of diseased vessels, as in the optic neuritis of the senile periods.

The microscopical appearances of optic neuritis are very uniform, and the opportunities of examining nerves in this state are not infrequent. Neuritis is by no means confined to the optic nerves, but is to be seen in many other nerves, both cranial and spinal. I have in my own possession¹³ sections of almost all the cranial nerves exhibiting neuritic change; the only differences between them rest upon the various degrees of richness in connective tissue, upon the rate of the neuritic or sclerotic process, and the amount of fatty degeneration of the nerve tubules and other products which accompany it. Changes of this kind have been described as occurring in the great nerves of the limbs, especially in cases of contraction of the limbs after paralysis, and after wounds or injuries. One of the most striking specimens of neuritis which I now call to mind, is a case of neuritis of the median nerve in the hand, which is shown in the Schröder van der Kolk collection in the Oxford Museum. In this most interesting case the hand had been amputated after mischief had been going on in the wrist for two years. The median nerve is thickened, irregular and sclerosed, and the neuritic process has *descended* as far as the digital branches. This tendency to propagate itself along the sheath of a nerve is very characteristic of neuritis, and it is by means of this property that neuritis occurring in any encephalic portion of the optic nerves, sooner or later presents itself in the eye. In the optic disk we find the sheath of the vessels much condensed and thickened, and evidences of interstitial inflammation in abundance. If we examine the sheath of the trunk, we find it full of proliferating nuclei and young cells of great instability. In the later periods the nerve columns may be seen to have wasted, and the connective tissue to be considerably increased. This sclerosis, which also affects the vessels of the disk, will,

¹³ I have also to thank Mr. Hulke for opportunities of observing some beautiful sections of optic neuritis prepared by himself. There is a good description of these pathological changes by Virchow in Gräfe's 'Archiv.' xii. 2, p. 117.

I think, explain the comparatively little congestion in neuritis. Iwanow has shown that 'perivasculitis' often extends for some distance along the main trunks, and is visible as a streaking along their course¹⁴.

The nerve is always affected in its entire thickness, and inflammation in one nerve invariably crosses over the chiasma, and involves the other likewise.

The cause of optic neuritis, when this depends upon encephalic changes, is meningitis or encephalitis. Optic neuritis does sometimes occur as an independent event of obscure causation, or it comes as a consequence of orbital mischief; but we have now only to deal with it as a symptom of head mischief, and as a symptom it signifies inflammation of some encephalic tissue, of membranes more commonly, of nerve masses less commonly. Optic neuritis is most common in those meningeal inflammations which, like the syphilitic, have the favourable conditions of contiguity, duration, and activity. A chronic meningitis lying at or near the base of the skull, and marked by active proliferation, is tolerably sure to set up optic neuritis. Inflammations which, like the tubercular, are contiguous to the optic tracts and nerves, and active in changes, but of short duration, cause optic neuritis as a fact, but not so constantly as a symptom, for the neuritis may not have time to reach the disks. As there is generally much exudation, and, moreover, much ventricular effusion with tubercular meningitis, we more frequently see hyperæmia and actual strangulation of the disks; or we see these phenomena at first, with optic neuritis as a later event. We do not see optic neuritis as a consequence of inflammations, such as traumatic meningitis, on the upper brain, as these inflammations, though active, and sometimes prolonged, are not contiguous. Ischæmia is more commonly the consequence of intracranial tumour than optic neuritis, and is due to pressure; optic neuritis does, however, occur no doubt as a symptom and consequence of tumour, and when it does so is due to meningitis or pos-

¹⁴ Cf. 'Iwanow über Perivasculitis,' in Zehender's 'Monatsheften,' Sept. 1865.

sibly to a belt of cerebritis which surrounds the tumour, and makes its way along the optic tracts or nerves. Unfortunately, almost all observers confuse optic neuritis with ischæmia, and we are unable from their words to say whether the disk was significant of disturbances of pressure or of disturbances of nutrition. From their descriptions, however, we may often see that what they call optic neuritis was really ischæmia papillæ. Mr. Hulke, on the other hand, has been very careful in preserving the distinction in the cases which he has recorded and classified. Those encephalic diseases which do not cause optic neuritis are sclerosis, hæmorrhage, and simple softenings of all kinds which are rather attended with atrophy. Abscess may cause optic neuritis in rare cases when it is of long duration, surrounded by widening irritations and in contiguous or connected parts. Periostitis I include in meningitis.

F. Chronic Optic Neuritis,

or 'red softening' of the optic nerve, is a term I wish to introduce as a name for certain lesser degrees of resistance to destruction which often precede white atrophy, and which are constantly overlooked. They are attended with congestive appearances, with very slight or, it may be, with no effusion, with feeble proliferation, and with but little disturbance of the central vessels. When I began to examine all cerebro-spinal cases with the mirror as a matter of routine, I quickly found that many states of the eye called 'simple progressive atrophy' had a distinctly hyperæmic stage, with sometimes a little effusion, preceding the whitening stage. This certainly has not been described in the eye-books generally, but I find it described by Mr. Hutchinson in the 'London Hospital Reports,' vol. i. In a very interesting paper on amaurosis, he says, in respect of the white atrophy, which he is disposed to attribute in some degree to tobacco smoking—'In this form of amaurosis the ophthalmoscope reveals to us remarkable and very constant conditions. In the early stage the optic disk is usually too red, and the whole of the choroid full of blood, and presenting the appearance of a pile of red velvet. There are no ecchymoses, nor any effusions of lymph.

In a little time the congestion of the optic disk diminishes ; instead of being too red, it is too white. At this stage the arteria centralis retinæ is much lessened in calibre, but its accompanying vein is of normal or even increased size. From this stage outwards the optic disk gets whiter and whiter, until all traces of arteries, excepting the larger trunks, are lost ; sometimes, but very rarely, even the largest cease to be visible. At this latest stage the veins are usually very small, but I have never known them absent. Whether the stage of preliminary congestion is always present or not, I do not know, since a large majority of cases come under notice long after it is passed.' I have little to add to this description by Mr. Hutchinson of a process which I have watched in all its stages in such cases as general paralysis. As to the hæmorrhage, I may say that the capillary congestion of the disk seems, in many cases at least, to break up into a minutely hæmorrhagic condition, the capillaries seeming to give way, and their contents to be extravasated, so that the carmine passes into browner shades, and the disk at the end of the congestive stages appears smudgy, or as if stippled with dirty brickdust. One sometimes sees these appearances likewise in the brain in cerebral softening. I have observed again, in many of these cases, effusion to slight but decided degrees—effusion distending and blurring the nerve, or even spreading beyond it. For instance, it was present in Case No. 105 of the Appendix—a case of 'locomotor ataxy,' in which the amaurosis is always supposed to be of the simplest progressive kind. In that case there was no complaint of loss of vision. This most uncertain symptom seldom sets in until atrophy is well forward, as we see also in general paralysis. To base any inferences, therefore, as to the states of the optic disks upon degrees of vision, is simply a waste of time.

Chronic neuritis or softening is not associated with perineuritis, that is, with extension of inflammation upon a belt of the retina. I have only once examined the structure of the optic nerve in the red stage of chronic neuritis, but in that case I did not find any marked proliferation of nuclei from

the neurilemma. I found dilatation of the capillaries, varicosity, and breaking up of the fibrils, a good deal of granular matter and some nuclei, probably from the interstitial connective tissue. We want, however, many more examinations of the nerves in this state. I have examined a very large number of nerves in full atrophy after chronic neuritis. The nerve fibres are generally destroyed, a few only perhaps remaining entire; the vessels are often degenerated or thickened, and the rest of the trunk is made up of granular matter, oil globules, colloid bodies, &c. There is, in fact, a degeneration, with very slight evidence of vasculo-cellular resistance. The causes of chronic neuritis remain very uncertain. It is not due to meningitis, as there is no ischæmia nor acute neuritis with implication of the sheath. I think tumour is always followed by ischæmia, by acute neuritis, or by simple atrophy due to disconnection from the optic centres, and not by chronic neuritis. Perhaps we may see chronic cerebritis around a tumour or clot propagated as chronic neuritis to the optic nerve, though I have not traced such a process.

Generally speaking, indeed, microscopic experience leads me to say, that whenever irritation is propagated along the optic nerves from inflamed brain or membranes, there is a proportionate degree of proliferation of the connective tissue, and I shall point out in my next chapter that chronic optic neuritis is best seen in the three kinds of sclerosis which are known clinically as general paralysis, locomotor ataxy, and palsy with tremor, and in the diseases allied and connected with these by processes which present intermediate characters.

G. *Retinitis*,

the condition which at the outset is marked by hyperæmia both of the disk and retina, and afterwards by the appearance of silvery patches of exudation upon the retina, is an affection too well known to need any description from me at present. Of course we must not confound this condition with the retina of albuminurics. In some ophthalmic works I have been surprised to find that retinitis 'is frequently the result of cerebral

disease.' Now, out of perhaps one thousand cases of cerebro-spinal disease, of which I have ophthalmic notes, I have found general parenchymatous retinitis in two only. In one of these cases, the retinitis was kindly watched for me at short intervals by my friend Mr. Oglesby. See Appendix, Case 39.

H. *Perineuritis*¹⁵.

I accept this name from Galezowski, though there is no real distinction between it and interstitial neuritis, in which there is always more or less perineuritis. In some cases, however, probably of slow change, the inflammation seems to affect the outer neurilemma more severely, and to extend over a wider belt of retina. I have not had such parts under the microscope, but I conceive that we should find great proliferation around the nerve and less change in the interstitial connective tissue. Galezowski, who alone has used the word perineuritis, says:—'The papilla is prominent and enlarged, but one readily sees with the ophthalmoscope that all the exudation is confined to the margin of the papilla, the outlines of which are veiled, while the central parts are transparent and more like the normal state. The capillaries are only developed at the periphery of the papilla; the central vessels are varicose, and sometimes bordered by an exudation.' He says this form is often confounded with the albuminuric neuro-retinitis, and this mistake might easily have been made in a case of scarlatinal kidney which Mr. Teale showed to me a few months ago. In the rest of his paragraph and in his pictures Galezowski is not so accurate, as he fails to distinguish between optic neuritis and ischæmia. His picture of perineuritis is more like neuritis, and his picture of neuritis is clearly from a case of ischæmia. I do not think there is much to be gained by the name 'perineuritis,' and prefer to include it in neuro-retinitis.

¹⁵ This section I leave as I wrote it, though I am tempted to cancel it. Physicians who use the ophthalmoscope will probably find, as I found, that our earliest tendencies are to multiply subdivisions, but subsequently to learn that such subdivisions do not correspond to any essential distinctions. Nay more, that ophthalmic appearances are so variable that it is difficult to make classes to include all cases.

I. *Consecutive Atrophy*¹⁶.

I will now describe the kind of atrophy in which ischæmia papillarum and optic neuritis may end. Dr. Hughlings Jackson has made popular a certain distinction between an atrophy following neuritis and an atrophy not preceded by neuritis. The former, or 'consecutive atrophy,' he would distinguish by the raggedness of its edges and the blurring of its outline; the other—'simple or progressive' atrophy—he would distinguish by its more brilliant appearance and by its clean-cut even rim. Galezowski, too, distinguishes consecutive atrophy: 'qu'elle est caractérisée par les contours irréguliers, frangés, mal limités, du disque optique,' etc., and the distinction is very commonly accepted among ophthalmic writers. Now, although this distinction is, or has been, valuable in drawing attention to the modes of atrophy, yet I think it is only partially true. The simple even atrophy often succeeds a chronic neuritic process, and, on the other hand, the ragged atrophy following acute neuritis sooner or later loses its blurred and irregular features, and settles down into the even staring state which, I think, is the end of all atrophies. The more I see of the histories of 'simple white' atrophies, the more I am assured that these states are often preceded by ischæmic or neuritic processes. In tubercular meningitis, for example, I have on my notes cases illustrative of every phase of the eye symptoms. In them I find that the most violent ischæmia or neuritis, which passes into the *transition atrophy* (as I would call it)—the ragged state with swollen retinal veins and exudative patches grouped about the margins—does also ultimately settle down into the even-edged and staring form. This kind of amaurosis, which I have found so often in blind asylums and among idiots, is not a mere degeneration signifying 'irritable' brain tissue or deficient development, but is a result of such causes as intra-uterine or infantile meningitis. After ischæmia, then, and after acute neuritis, the disks no doubt present distinctive features during a longer or a shorter time, according to the degree of effusion or of neuritic

¹⁶ Vide Liebreich, 'Atlas,' Tab. xi. figs. 13 and 14.

resistance—this is *transition atrophy*. The swollen disk recedes gradually, and the reds and greys give place to dirty white, the margins being either completely blurred, or slowly extricating themselves here and there, as if the disk had been crushed and its contents squeezed out. Streaks of exudation, which are often continuous with smudgy parts of the disk, remain, though not so long, in the course of the retinal vessels. Little by little the disk clears up; it whitens and the edges become more detached, the exudation often remaining in little grey satellitic dots around it. All hæmorrhages shrivel, whiten, and disappear, and the fine vessels are no longer seen on the face of the disk, though there is not the same delicate shrivelling and waning of them against the white background which we see in chronic neuritis. On the retina the arteries, which had long been diminished in size, remain small, while the dark thick tortuous veins decrease slowly. As the face of the disk clears and whitens, they diminish a great deal, though they never sink much below the normal standard. We find, on examining such nerves with the microscope, that the new but instable connective elements disappear slowly even after great inflammatory proliferation, until the nerve becomes a mere fibrous cord, as in primary atrophy. Very commonly ischæmia does not result in complete atrophy; and I have some reason to hope that neuritis does not always, for under the microscope I have several times seen advanced neuritis and great proliferation with the nervous fibres still intact or almost intact; but I have always believed that neuritis means great danger within the head. I have now several little patients under observation, who, having suffered from meningitis with ischæmia papillaris, are passing through the peril of atrophy with good hopes of safe vision. The danger of meningitis to the sight has, however, been known for a great number of years. Galezowski thinks that consecutive atrophy admits of melioration and cure in perhaps one case out of every three. But these statistics are of less value, as he has not recognised the distinction between ischæmia and descending neuritis, which latter is far more dangerous to vision. Mr. Oglesby has given a good deal of attention to the final results of atrophy

in their bearing upon vision, and he has recorded¹⁷ some very interesting cases of atrophy of the second and third degree, in which some sight was regained after long lapses of time. Like Galezowski, however, Mr. Oglesby has not clearly distinguished in the histories of his cases between consecutive and primary atrophy; nor, again, between the two kinds of consecutive atrophy—that which follows ischæmia papillaris, and is probably confined to the disk, and that which follows descending neuritis, and is a wasting of the nerve in more or less of its length. When old amaurotic cases present themselves for treatment, I believe it is often impossible to tell, by the ophthalmoscope alone, to which of these classes the atrophy belongs.

I need scarcely say that these modes of atrophy must be distinguished from—

1. Atrophy with (great) excavation or glaucoma. This is known by tension of the eyeball with ciliary pain, by the double border of the disk (the choroidal border and the edge of the cup), by the shadow thrown into the cup, by the curious incurvation of the vessels, by the atrophy of the neighbouring choroid, &c. In cerebral atrophy, if the cup be rather deep, it is never abrupt at the edges.

2. Atrophy following 'pigmentary retinitis.' Here the mischief is mainly and firstly retinal, and the retinal vessels are diminished.

3. Atrophy following syphilitic or other irido-choroiditis. They are attended with widespread retinal mischief, and do not at all resemble cerebral atrophies.

4. Atrophy following myopia. Is seen only in extreme cases, and then with posterior staphyloma.

5. Atrophy following albuminuric degeneration. Is distinguished by the peripapillary and retinal distribution of the mischief, by the presence of fresh or old hæmorrhages, and of fatty patches; also by presence of albumen in the urine, &c. This atrophy generally follows, or always follows, extensive destruction of the retina, but I have seen some early

¹⁷ 'Lancet,' Aug. 22, 1868. Also 'Royal Ophth. Hosp. Reports,' vol. vi. pt. iii. 1869.

cases where there was little more than a pæony-red tint around the disk, and some hyperæmia, and these might mislead even a wary observer, unless all the symptoms were investigated.

K. *Primary Atrophy.*

Atrophy which is 'primary' at the disk may nevertheless be the consequence of neuritis, for neuritis in some intracranial part of the nerve length may reveal itself at the disk simply as atrophy—as atrophy due to a separation of the distal from the central parts. Atrophy presents two different aspects to the observer, though I shall not dwell long on the difference, as it seems to be of little clinical importance. The one kind is a grey shrinking of the nerve and its expansion, with a great tendency to cupping; the other is a dense tendinous white disk with a good deal of connective tissue growth, and less tendency to cupping. The former kind involves only the filament and ganglion layers of the surrounding retina, while the latter invades all the layers, and is probably always preceded by some degree of neuritis, acute or chronic. In both cases the finer vessels fade away, leaving the disk a dead white, and in dense atrophy the thickening and contraction of the sheath of the central vessels lessens even these in calibre. Under the microscope we find, after all the more active periods of degenerative hypertrophy have passed away, that neither nervous elements, nor perhaps any fatty and granular remnants of them, are anywhere to be seen. Even the new connective elements themselves dissolve and shrink down into a mere band of fibrous tissue, containing the tracks of degenerated vessels and a few wrecks of bygone structures, cellular and other. These changes largely involve the retina also, though it is surprising how little central vision may be affected in the earlier stages, or in the not uncommon incomplete forms. I used to be amazed sometimes to see white and even glistening optic atrophy in eyes which could read ordinary types with ease. Such cases are only discovered by the physician, at any rate in their earlier stages, and sometimes the mischief seems arrested at these earlier stages. On the other hand,

sight sometimes seems to vanish when the mirror betrays but little whitening or apparent degeneration. In the former cases some nerve fibres must survive much longer. Primary atrophy also may occur in any part of the nerve length from the quadrigeminal bodies downwards, and so affect vision very seriously before the disks are actually reached.

Of the diagnosis of atrophy I have said something already in the section on anæmia. It is often very hard to tell whether atrophy be or be not present in the early stages. Mapping the field will help us, for if the deficiency of vision be uniform, we have probably a state of anæmia present, and anæmia only; if, however, any part of the visual field be wanting, we have to fear that the state is one of commencing atrophy. In simple atrophy, which is more dangerous to vision than consecutive atrophy, we have a more evident dwindling of the vessels. The vessels often begin to shrink a little from the first, and when they do we have an important sign of atrophy before us.

Or we may detect a slight cupping, which is a sign of atrophy, but which may unfortunately also be a physiological peculiarity. Cupping, as an unimportant abnormality, has been described in the third chapter; I will only add here a few remarks on the cupping of atrophy. In the first place, let me say that it is scarcely possible for the observer to make much of an atrophic excavation in the inverted image. In the erect image we see a uniform depression with sloping sides, not thrusting out the lamina cribrosa, but simply showing a thinning away of substance from before it, and allowing the pitting of the lamina to become more visible¹⁸.

The course of the vessels must now be very minutely regarded. By slightly varying the lenses, the observer may satisfy himself of the degree of excavation, and may tell whether the vessels do or do not bend sharply over the edge of the basin. If there be an abrupt bend as they dip, then the case is one of glaucomatous excavation; if

¹⁸ In consecutive atrophy the disk may be prominent with the products of previous cell growth not yet dissolved, or it may have a fictitious prominence due to the wasting and subsidence of the surrounding belt of retina.

the bend is quite gradual, the excavation is not glaucomatous. A binocular instrument is useful in these cases, but unfortunately it is only available for the small inverted image.

The causes of simple atrophy are of four kinds. Firstly, it may be due to any cause, such as a tumour, or a patch of inflammation in the course of the nerve behind the eye, which severs the nerve fibres and leaves the separated parts to wither. Secondly, it may be due to destruction of the centres of vision making the nerve useless, this being merely another form of the first cause. Thirdly, and very commonly, it is a sclerotic process associated or continuous with patches of sclerosis elsewhere, as in the spinal cord in locomotor ataxy, or in the encephalon in several forms of chronic degeneration. Fourthly, it is often due to deficiency of nutrition, such as we see in senile degeneration of the arteries, in embolism and the like. Atrophy due to arterial failure and suggestive of general senility in the encephalic arterial system is very valuable as a symptom, but is often so slight as to be doubtful, or to be mistaken for the normal grey of age. When it occurs it may be associated with central softening and hæmorrhage.

In concluding this section I should add that certain curious discolorations of the disk sometimes precede atrophy. They may be seen in the inverted image when they are far advanced, but in the erect image are often to be seen much earlier. We sometimes see a greenish and sometimes a bluish tint. The word 'slaty' may express the tint, some slates being greenish, others rather blue. This may be some stage of chronic neuritis, a sort of ecchymotic change, or again it may be sclerosis which gives the same grey look to the optic nerve that it gives to the pons or to the columns of the cord. I have certainly seen it sometimes follow a red disk. It is not uncommon in symptomatic nerve disorders, in encephalic or spinal disease, and not uncommon again in the amauroses of lead and other slow poisons. In rare cases we may see one disk in evident atrophy, while the other is curiously discoloured. One or two pictures of such disks are shown in Liebreich's '*Atlas*,' Plate xi.

CHAPTER V.

ON THE RELATIONS BETWEEN CERTAIN INTRACRANIAL DISORDERS AND AFFECTIONS OF THE OPTIC NERVE AND RETINA.

THE most important question which presents itself to us in this part of our enquiry is the general question—does degeneration of the optic nerves always mean that there is some serious lesion in the cerebrospinal cavity?

In dealing with this question I must ask leave to set aside, as foreign to the main object of my investigation, all those causes of change in the optic nerve which are confined to the orbit and face, or which originate at least without the encephalic cavity. This very interesting class of cases belongs rather to surgery than to medicine, and has been well described by many writers, especially by Mr. Salter in his paper on 'Nervous Affections from Diseases of the Teeth,' in the Report of Guy's Hospital for the year 1868.

Now, these exceptions being made, and likewise all albuminuric, glaucomatous and other merely local changes being classed apart, what are we to infer from the presence of papillary ischæmia, optic neuritis, or optic atrophy? Up to a quite recent time, I believed that the existence of such changes pointed almost certainly to cerebrospinal mischief. Of late, however, my opinion has become less strong, and I have begun to think that I assumed too close a connection between optic neuritis and cerebral mischief. In preparing to write this volume, I have run through a great deal of the

writing of ophthalmic surgeons, and I have read notes of a large number of cases of neuritis and of atrophic amaurosis in which there seemed to be little likelihood of concurrent cerebral disorder. When an ophthalmic surgeon records cases of optic neuritis as occurring in persons who present no symptom of cerebral disorder, and records them side by side with other cases in which cerebral disorder undoubtedly existed, showing thereby that he was fully alive to the probability of the co-existence in both classes of cases,—when, moreover, these persons present themselves time after time for examination and develop no symptoms beyond those of the eye, and go through all the local changes of neuritis followed by atrophy,—when we see such cases carefully recorded, as they are by Mr. Hulke, for instance, in his interesting notes published in the ‘Royal Ophthalmic Hospital Reports,’ vol. vi. April, 1868, we physicians must, I think, be prepared to modify our previous exclusive notions, and to admit that there is no necessary connection between optic neuritis and cerebral disease on the one hand, as the surgeon, on the other hand, admits that cerebral disease may exist without optic neuritis¹.

At the same time, after making full allowance for the different conditions under which ophthalmic surgeons and physicians are practising, remembering also that no patients come to a physician for eye mischief alone, yet I cannot but think that uncomplicated optic neuritis is a rare event. From the very nature of the circumstances, when we have a case of optic neuritis before us, we are only able to base our supposition of a sound encephalic condition upon negative evidence. A small morbid growth or inflammation so placed as to interfere with the optic nerves or centres might, for a long time at least, give rise to very few symptoms. I have a patient under my care at present with well-marked symptoms of cerebral tumour, who had been afflicted with amaurosis from atrophy for three years before any cerebral symptoms

¹ A similar local neuritis may account for the occasional appearance of palsy of one orbital nerve independently of syphilis, locomotor ataxy, &c.

became manifest. Again, as in Case 77, a patient may have suffered from cerebral symptoms in time past, but which have wholly or partially subsided: he may then begin to suffer from loss of sight, and seek the assistance of an ophthalmic surgeon, and the surgeon may not light upon the old history. In Case 78, the history of old cerebral symptoms had almost escaped me, who am constantly on the look-out for such things, and the woman only recalled them by an effort of memory. The cerebral symptoms of Case 77 might easily have escaped notice, as the only symptom the woman complained of was 'sick headache.' Here we are met by the grave difficulty which besets all new clinical investigations,—we have no long list of autopsies to check or inform our judgments. Cerebral diseases are often so long in their course, and pass under so many medical men in turn, that the uncertainty of obtaining post-mortem examinations, which is great in all cases, is greater than ever in that class with which we are now dealing.

It is sufficient, at present, to say that the occurrence of papillary ischæmia or of optic neuritis in any person ought to awaken the gravest suspicion of cerebral disorder, and that the occurrence of simple atrophy should suggest great watchfulness; that the precise degree of meaning to be attached by the physician to optic neuritis cannot, however, be determined until a certain number of cases of optic neuritis, without apparent cerebral changes, have been traced to the post-mortem table. Meanwhile, the physician will not often be called upon to determine the meaning of neuritis alone, it will come before him in cases where other symptoms will also be present to assist his diagnosis, and we must follow up the uncomplicated cases at eye hospitals.

I shall now discuss the effects of various forms of encephalic disease upon the optic disk and retina. I propose to take these various forms separately, and to ascertain what changes in the optic nerves are known to accompany each of them; we shall then be in a position to try to draw some general conclusion concerning the way in which encephalic

changes are propagated to the optic nerves, and concerning the importance of the messages which these nerves convey to us. I need only look to convenience in making a classification of encephalic disorders; the following arrangement is, I think, defensible from that point of view, and that only:—

- I. Epilepsy, page 77.
- II. Chorea, page 83.
- III. Mania, page 83.
- IV. Dementia, page 85.
- V. Meningitis, page 85.
 - A. Tubercular. B. In Pyæmia and Fevers.
 - C. Syphilitic and Rheumatic. D. Of Drunkards.
 - E. Traumatic.
- VI. Concussion and Fracture, page 109.
- VII. Hydrocephalus, page 112.
- VIII. Tumours and Periostitis chronica, page 114.
- IX. Atheroma, Softening, and Hæmorrhage, page 178.
- X. Cerebritis, Abscess, and Sclerosis, page 186.
- XI. General Paralysis, page 193.

I take a disease first which is very interesting from its almost negative results as regards the eye, so that, before seeing what sort of diseases do permanently affect the eye, we may see that some very serious diseases have no such consequence.

I. *EPILEPSY*

is a name which we are not to apply to convulsive symptoms generally, but to a certain group of convulsions which tend to periodical recurrence, affect both sides of the body, are closely associated with unconsciousness, and do not depend upon obvious disease of the encephalon. So long as we are to use the word ‘epilepsy’ at all, let us use it in this rigidly defined sense, as laid down by Dr. Russell Reynolds; in no other sense can epilepsy be appropriately said to name a disease, for it can have no other constant

application. It is generally held, at present, that the convulsions in which epilepsy almost wholly consists, and which form a part of many other diseases, such as encephalic tumour, or meningitis, depend upon partial anæmia of the hemispheres and central ganglia. Such convulsions occur in animals which are bled to death. In epilepsy they are supposed to depend mediately upon a spasmodic contraction of an arterial region, such, for instance, as the region of the middle cerebral artery². This hypothesis seems to be supported by many observations, but as yet is very far from proof. Among the analogies which favour it may be named the sudden losses of equilibrium which seem to occur in other arterial regions, as in the anterior lobes and olfactory lobes of the encephalon, and as in the retina. Diarrhœa, diuresis, and many other temporary disturbances of function are often traced with much likelihood to the influence of nerves upon vascular supply. We have also arrived at similar conclusions, experimentally, from researches made into the influence of nerves upon the vascularity and so upon the function of the salivary glands, upon the nutrition of the eyeball, and so on. In epilepsy, the sudden pallor of the face is commonly quoted as an evidence of cerebral pallor; little weight, however, can be attached to this observation. In the first place, it is not constant; and secondly, it does not appear to be generally noticed that the arterioles of the surface of the head are under the same nervous governance as those of the brain. The pallor of the face is more likely to be due to the general nervous shock, and is akin to the pallor which accompanies other nervous disturbances, which are set up either from within or from without by such influences as terror and the like³. I am myself disposed to

² Vide Dr. Hughlings Jackson, *passim*.

³ A great number of epileptics complain of disordered vision before or after their fits, but this is often the effect also of general nervous shock; it is transient, and it coincides with disorders of other functions governed by the brain, such as perturbed hearing and speech, vertigo, and the like. I have never been able to trace it to any indubitable change in the disks or retinas, though I have over and over again examined the eyes under these circumstances. Vide cases in the Appendix.

attach great weight to the observations and reasonings which have attributed epilepsy to disturbances near the fourth ventricle. This is not the place to enter upon so large and so interesting an enquiry, but I will only refer to the well-known occurrence of bilateral convulsions exactly like epilepsy, which follow an injury below the floor of the fourth ventricle. This experiment has been recently repeated and verified by Dr. Nothnagel [Virchow, 'Archiv.' vol. xliv. 1868], who determines with great minuteness the precise spot whence these convulsions are determined. Whether the convulsions of epilepsy be due to disordered blood-supply in the great central ganglia, or to some disturbance, vascular or other, at a 'cramp point' near the fourth ventricle, is therefore a very interesting subject for enquiry. The little evidence which we receive from the ophthalmoscope tends rather to favour the otherwise likely supposition that epilepsy is the common result of more than one cause—that not 'symptomatic convulsions' only, but 'genuine' bilateral tongue-biting fits are either the way in which the whole encephalon manifests an overthrow, which, again, may be variously brought about, or may be in other cases the consequence of a local disturbance, say, in the pons, or upper medulla oblongata. For instance, my own experience of post mortems in epilepsy would lead me to think that venous congestion may have the same effect upon cerebral function as anæmia, and the ophthalmoscope points to a like conclusion, for I have noticed congestive appearances in the optic disks more than once in persons suffering from epileptic fits. The indications of the ophthalmoscope divide themselves naturally into two parts; into the appearances seen during the attacks, and those seen during the intervals between the attacks. The difficulty of examining a patient during an epileptic fit is considerable: sometimes, however, it may be overcome with a little dexterity and patience. I have managed to see the disks distinctly during the convulsions in many cases of epilepsy, and of six cases I have careful descriptions in writing. [Vide App. Nos. 2, 3, 5, 6, 8, 17.] In three of these I found an anæmic condition

of the disks, and in three a hyperæmic, or congested condition; a curious opposition of experiences. Of the anæmic cases two were seen in the Leeds Infirmary, and one in the Wakefield Asylum: those seen in the infirmary were both out-patients, who were dæmonized during the hour of attendance. In these two I saw of course the character of the convulsions, and knew the past history of each case. In both, the disks were very white and small, though in both the arteries could be distinguished from the veins; the whiteness trespassed to a small extent upon the surrounding belt of retina. I examined the eyes of both the patients before and after the day of the convulsions, and noted nothing, or but little abnormal. The third case, which I saw at the Wakefield Asylum, is the young man C. W——, marked No. 13 in my list of cases of epilepsy with insanity, published in the 'Transactions of the Medical and Chirurgical Society' for 1868, and republished in the Appendix.

On one of my visits I had examined his eyes, and noted the disks as healthy, the vessels being large and full. On a subsequent visit he was brought to me in haste, by Dr. Crichton Browne, as he had just passed through a violent epileptic paroxysm, and still had visual and other hallucinations. I found the 'vessels very small, few and fine,' in the right eye; in the left, 'vessels smaller than before, but artery and vein distinctly visible.' Dr. Hughlings Jackson has reported more than one case in which he examined the optic disks during epileptic states, and found them to be anæmic; one very interesting case was published by him in the 'Medical Times and Gazette,' for Oct. 3, 1863, and is reprinted in the Appendix, No. 2.

Mr. Carter once told me that he had examined the eyes of a patient who had just passed through an epileptic paroxysm. He saw the optic disks as soon as convulsion ceased, and found that the nerve tissue was almost as white as in atrophy. The state of the retinal vessels he had forgotten. The number of observations of the optic nerves at or about the times of convulsion in epilepsy is not great, the occasions of them are rare, and the difficulties considerable; but it seems so far

that there is a certain agreement in the results of those which are recorded, and that the evidence in favour of a very decided anæmia of the optic disks being a frequent, if not a constant, phenomenon during the convulsions of epilepsy, is quite worthy of attention. If it be true, it is a very remarkable and important discovery. I am disposed to think that the optic anæmia may persist for some hours, or even perhaps days, after the convulsion has passed away. In some of my own cases, in the case published by Dr. Jackson, and in Mr. Carter's case, the whiteness was noted after the convulsions had passed away. I may refer also to Case No. 6 in the Appendix, as showing how long some anæmia of the disks may survive the convulsions. This case is not included in the half-dozen to which I have already made reference. I shall return to this question when I discuss the state of the disks and retinas between the paroxysms. Before leaving the present subject, however, I must still point out that anæmia of the optic disks has not been by any means a constant condition in my observations during epileptic convulsions. I have said that in three out of six cases I found decided hyperæmia with more or less exudation in the disks. Case No. 5 is one of these, and I publish it because we proved, by post-mortem examination, that there was no 'coarse disease' in the brain. All three were, however, cases of prolonged convulsion alternating with times of stupor—of the *status epilepticus* as it has been called. In these three cases I found the disks greyish red, and perhaps a little swollen; the borders were somewhat concealed, and the retinal vessels were very dark and full. In the case which we examined after death there was great venous congestion of the encephalon; and I have no doubt that the state of the disks was one of slight ischæmia, and was due to congestion in the cavernous sinus. The state of the disks varied little or not at all during the change from convulsion to stupor, in each case; nor did the retinal vessels vary in the least with the convulsions of the iris. These observations support my belief that convulsions may accompany venous hyperæmia of the brain as well as anæmia, both conditions being suppressions of arterial supply; at any rate, they must not

be lost sight of in any comprehensive discussion of the subject.

During the intervals it may be said, in a general sense, that the optic disks and retinas regain their equilibrium and appear normal. There is usually no appearance during the intervals of epilepsy proper which would justify an ordinary or a casual observer in pronouncing that the back of the eye is otherwise than healthy; nor is there any permanent disturbance of sight in those cases which are attended with periodic amblyopia. After a large experience of epileptic eyes, however, and after careful comparison of them as a class with the average state of the eyes in healthy persons, I think I am justified in expressing an opinion that in epileptics who suffer from violent fits, or from frequent fits, there is during the times of repose a higher degree of vascularity in the optic disks and retinas than is quite normal. There is often a pronounced redness in the disks of such patients, which does not obscure their edges, for it is attended with no exudation, but which makes the white centres shine out with unusual brightness. The retinal vessels in such cases also are large, both the arteries and the veins being full, and the minuter branches of both can be traced more easily in their course in the retina.

I have been accustomed for some time to point this out to my colleagues and pupils, and have often said that I thought I could pick out an epileptic, by the appearance of his disks and retinas, from a row of healthy persons. I have recently noticed that M. Bouchut expresses the same opinion. I think M. Bouchut is often rather hasty in pronouncing upon degrees of vascularity in disks and retinas, but in the present case I am prepared to give him a general support. If it be finally agreed that such appearances are common in the eyes of epileptics during the intervals of health, we shall have discovered a fact which is of high interest, when taken together with the changes noted during the attacks; for it may signify either a general vascular relaxation, or a venous congestion secondary to frequent arterial disturbances.

Optic neuritis, ischæmia papillæ, and atrophy seem to be

as rare in epilepsy as they are common in symptomatic convulsions.

II. CHOREA.

The eyes of patients suffering from chorea have been examined with the mirror in a large number of cases by Dr. Hughlings Jackson and myself, and no doubt by many other investigators.

It may be looked upon as established that no change either in the vascularity or in the nervous tissue of the disk and retina is to be found in simple chorea. I have not thought it necessary to publish any cases in support of this statement, though I do publish one case in exception which was reported by Dr. Hughlings Jackson (Appendix, No. 19); it is interesting to notice that the signs resembled those sometimes seen in cases of undoubted embolism.

III. MANIA.

Mania which depends upon such marked anatomical changes as meningitis and the like is not here to be considered, but only that form of acute insanity in which the pia mater and hemispheres are found to be highly hyperæmic. As some forms of acute melancholia attended with great variety of action nearly approach mania both in symptoms and in the post-mortem appearances, the subject of this paragraph may be stated as acute insanity with cerebral hyperæmia. In that form of melancholia which it is better to call 'profound' rather than 'acute,' and which depends less upon hyperæmia than upon serous exudation, and perhaps upon anæmia, the fundus seems to be either unaffected, as I have shown in a contribution to the 'Medico-Chirurgical Transactions' of 1868, reprinted in the Appendix; or, if affected, is but simply anæmic, like the rest of the tissues in the body. In one or two cases of recent and profound melancholia I have notes of reddish disks, but I cannot attach much importance to them. In acute insanity, however, I have shown to some extent in the same paper, and have since had occasion to

notice, that during a short but variable time after the paroxysm, the back of the eye, when examined with the mirror, presents a vascular suffusion or pinkness—a pinkness so great, after severe paroxysms, as to obscure the disks. A like suffusion is sometimes to be seen also in the conjunctiva. No exudation or permanent mischief is seen unless the mania be complicated with some more definite structural change in the encephalon. The difficulty of seeing the disk is at least as great in maniacal paroxysms as in epilepsy, generally perhaps much greater. It is not often pleasant to try to examine the optic disks of a raving maniac, and I have only once seen the disks in such a case. In that one instance I found the disks white: the patient, B. W. S., age 23, in the Wakefield Asylum [Schedule II., No. 31, Appendix], was suffering from acute mania, and by good fortune we secured an examination during a paroxysm. The right fundus was anæmic and the disk rather white, looking like atrophy: the left eye was like the right, except that there seemed to be some normal vascularity at one point of the margin of the disk. I cannot attach much weight to this single observation, but I may allude to it as suggesting that mania, like epilepsy, may be due to vascular spasm.

My observations of the hyperæmia of the eye after the paroxysms correspond closely with the state of hyperæmia of the brain noticed after death in such cases. Should my statements be verified hereafter, either by myself or by other more competent observers, the state of the disks and retinal vessels in mania will be a remarkable and valuable proof of the close dependence of their circulation upon that of the brain, and will add much to our confidence in reasoning from the one to the other. As a means of diagnosis it will be seen that the use of the eye mirror in the investigation of insanity is chiefly for the distinction of 'organic' from 'functional' disease. As in epilepsy, when the mania is symptomatic of 'coarse disease,' we find permanent changes in the disks due either to obstruction to the intracranial circulation, that is, to ischæmia, followed perhaps by atrophy, or due to neuritis, or again, to simple progressive atrophy. No such changes

occur in 'functional mania.' [Vide Cases of Mania in Appendix.]

IV. *DEMENTIA.*

In dementia the changes seen by the ophthalmoscope are numerous. I found marked changes in and about the optic disks in twenty-three cases out of thirty-eight: six more being recorded as doubtful. [Vide Appendix.]

None of these cases were epileptics, but the class of dementia is a very heterogeneous one, and includes worn-out lunatics of all sorts. A large number of those whom I examined were known to have organic disease of the encephalon, and this accounts for the high average of optic disorders among them. We shall gain nothing, I think, by associating optic changes with dementia as such; so far as these are the consequences of organic disease, we shall discuss them in other paragraphs with more perspicuity.

V. *MENINGITIS.*

A. Tubercular Meningitis; or Granular Basilarmeningitis.

In some forms of encephalic disorder—as in chorea, for instance—the ophthalmoscope gives us little or no help; in other forms—as, for example, in general paralysis or encephalic tumour—its discoveries have important pathological meanings; in other forms, again, the ophthalmoscope may give us most valuable help in diagnosis, and this is the case in some kinds of meningitis. During the last few years I have given a great deal of time and care to the investigation of the states of the eye in meningitis, and I hope to be able to show that my pains have not been thrown away. I shall first speak of that form of meningitis which is called 'tubercular,' for it is upon this form that I have the most important statements to make.

I began to use the ophthalmoscope in meningitis as a help to the general study of that most important disease. I had long been convinced that tubercular meningitis, as a disease of children, is more common and less uniformly fatal than is

generally supposed. In its milder forms, however, it is very difficult of diagnosis; and as before the discovery of the stethoscope the diseases called phthisis were supposed to be almost invariably fatal, because the cases which were so well marked as not to admit of doubt were fatal, while the nature of milder cases which admitted of recovery were open to question, so it is now with tubercular meningitis. Those cases which present no difficulty of diagnosis are the extreme forms which kill, while those which admit of recovery are not to be diagnosed with certainty. Can the ophthalmoscope help us in this difficulty, and may we hope to see it play in tubercular meningitis the part which is played by the stethoscope in tubercular pneumonia? The help we get from the stethoscope is this:—A patient who has been languid for some months, who has lost weight, whose appetite and digestion have been uncertain, and who may have spit a little blood, or have coughed a little, comes for our opinion. We then examine the chest with the stethoscope. We may find nothing, not infrequently we do find nothing, and in such cases we cannot go beyond a guess in our diagnosis, although the subsequent course of events may prove that the lungs were actually diseased. Or, again, the stethoscope may put us in possession of evidence which establishes the diagnosis, and enables us to ascertain that the lungs are diseased—a certainty which is not to be shaken by any subsequent course of events, even if they terminate in recovery.

Now I think I am in a position to say that we get some such help as this from the eye mirror in tubercular meningitis; that the mirror enables us to learn something more concerning the clinical history of meningitis than we know already, and that this additional knowledge will bring about some change in our views and opinions concerning the frequency and the curability of this formidable disease.

I have already described the modes of origin of ischæmia papillarum, of neuro-retinitis, and of atrophy. I need not again go over this ground to show that in meningitis the exudation at the base of the brain may press upon, or the inflammation may involve, the cavernous sinus, and slacken

the ebb of the blood, in which cases we have ischæmia only. Or the inflammation may creep down the nerve and cause neuritis optici, or may creep mainly along the sheath and cause perineuritis. Or, again, it may both interfere with the sinus, and so with the ophthalmic vein, and may likewise creep down the nerve; in such a case we should have ischæmia followed by neuritis. Finally, these states may be followed, and often, though by no means always, are followed, by atrophy; but it is not probable that simple severance of the continuity of the nerves often occurs in meningitis so as to cause a primary atrophy of the disks—an atrophy, that is, without forerunning congestion or inflammation. The anterior subarachnoid space, with the Sylvian fissure, is a district which, as we know, is especially one of the districts of tubercular meningitis, and the chiasma seldom escapes. In their backward course, from the chiasma up to the tubercula quadrigemina, we know also that the tracts are closely invested by the highly vascular pia mater, and that they are in great measure dependent upon it for their nutrition. Inflammation of the membranes upon the tracts, chiasma, or optic nerves, therefore, not only creeps up to the eye and presents its characteristic cell proliferation, but it also cuts off nutriment from the nervules. This compound mode of change I have several times verified with the microscope, and have seen the gross connective tissue of neuritis enclosing, not crushed nerve-filaments, but empty or half-empty spaces from which the filaments were withering or had withered away. This condition is best seen in some more chronic cases.

It is, I believe, the opinion of most if not all physicians, that tubercular meningitis is invariably fatal. I see, for instance, that Dr. Wilks, in his recent lectures on Diseases of the Nervous System, repeats and adopts this opinion. Many medical men, however, while upholding this belief, will nevertheless say that in the course of their practice they have seen one or two cases much resembling tubercular meningitis which recovered. The mere fact of the recovery makes them doubt their own diagnosis, and makes them suppose that the case which, had it been fatal, would unhesitatingly have been

called tubercular meningitis, cannot have been meningitis because recovery took place. Indeed, it is well known that the diagnosis of tubercular meningitis, even in well-marked cases, may often be very doubtful. Children not infrequently present symptoms much like those of meningitis, but which turn out to be significant of mere disturbances, or of cerebral diseases of other forms. And as this is true of cases which present decided symptoms, so much more is it true of cases presenting indefinite symptoms. The fever, the headache, the scream, the vomiting, the perturbed sleep, the strabismus, and other decided symptoms, followed by death, point undoubtedly to tubercular meningitis, and to an extreme case. But does meningitis occur only in extreme forms? or may it occur in milder forms which end in recovery? In milder forms we might have occasional but not urgent vomiting, some pain in the head but not violent pain, some startings in the sleep, or even slight passing convulsive attacks; but all these taken together may well be attributed to some disorder far less terrible than tubercular meningitis. Hence, as I have said, it appears that, at present, meningitis can only be diagnosed with certainty, or, indeed, with any great degree of probability, in its extreme forms, which extreme forms are of course largely fatal.

The important question for us to decide is, whether we have any means of detecting with certainty the presence of meningitis in those slighter cases where we can now only guess at it, or can scarcely even guess; and in which cases we need not expect to find a large percentage of mortality. It is here, I think, that the ophthalmoscope comes to our assistance, and gives us the same kind of help in detecting incipient or slight degrees of tubercular meningitis that the stethoscope gives us in detecting those incipient or slight degrees of ulcerative change in the lungs, which without it are beyond certain diagnosis. When a patient is seized with vomiting, headache, convulsions, and other symptoms of much meningitis, and when at the same time, on examination with the ophthalmoscope, I find congestion of the optic disk and

retinal vessels, which is frequently the case, then I have no hesitation in saying that the patient is suffering from meningitis at the base of the brain, and the autopsy proves the diagnosis to be correct. Suppose, however, that a child is liable to occasional vomiting of a 'purposeless' kind, and attended with but little nausea; suppose him to be liable to an evening fever, and to be rather restless, or sometimes very restless at nights; suppose, moreover, that he complains of pain in the head from time to time of a kind which drives him for a few hours only from his companions and his games, or perhaps for a day or two; suppose, again, that his temper changes, and from being a good child he becomes irritable or even positively mischievous, while at the same time his memory does not develop, and he is quite unable to fix his attention upon his school work; suppose, farther, that he suffers from spasmodic movements during his sleep, or even from full convulsions, and that the child nevertheless recovers from this state, and returns either to full health, or to health of body with more or less injured mental faculties,—should we call such a case meningitis? Now, I have had many such cases under my care, and I have records of many in which the ophthalmoscopic appearances were noted throughout, and in which I found those same signs in very well marked degrees, which I have also described as occurring in undoubted cases of meningitis, proved by autopsy. Is it not probable that in these cases we have also meningitis—a meningitis less severe than that which proves fatal, but meningitis nevertheless? The main difficulty I have to contend with in supporting my view of these cases is, that by the very nature of them I am shut out from any appeal to the post-mortem table. It may one day happen that some patient of mine, who has passed through these states of ill-health, may die from other causes, and give me the opportunity of verifying or disproving my hypothesis; but as yet no such opportunity has presented itself⁴.

⁴ These remarks were first published in the '*Lancet*' for May 1, 1869. In the same paper is described a case of supposed 'strumous meningitis' ending in recovery. The case was under Dr. Radcliffe's care. 'The right optic disk,'

I will relate one case, however, which fulfils these conditions to some extent—a case in which I diagnosed meningitis in a first attack, and the child recovered, but died from a subsequent attack.

Master O——, aged six years, was under the care of Mr. Mann, of Leeds, for obscure head symptoms, suggestive of meningitis. At the same time (1868) I was seeing another boy, Master R——, also under Mr. Mann's care, for similar symptoms. A few days afterwards I was requested to see Master O—— likewise, so that I followed the two cases together with Mr. Mann throughout their course. Both boys complained of fitful headache; intolerance of light; occasional purposeless vomiting, sometimes severe; much restlessness and starting, and slight occasional attacks of a more convulsive character. Now in both these boys I found with the mirror the condition I have described as *ischæmia papillæ*, and I accordingly diagnosed meningitis in both. Both the boys recovered in a few weeks, and I saw them on several occasions subsequently in my own house, where I also made repeated examinations of the eyes. The *ischæmia* slowly subsided, leaving a whitish look about the disks, which threatened in Master R—— to become actual atrophy; but the danger subsided, and the sight remained good so long as I had him under my notice. His recovery continues, though his mother stated when I last saw him that he still suffered from some mental incapacity, and a little irritability of temper. In the case of Master O——, the *ischæmia* of the disks likewise slowly subsided. He walked several times to my house with his mother, a distance of at least a mile, and he joined in the sports of his fellow-boys and girls. One day about six months later he came into the house complaining of his head, and the old symptoms of meningitis returned. I again saw him with Mr. Mann, and discovered neuro-retinitis. In this attack he died, and we obtained a post-mortem examination. We found meningitis, and the

it is said, 'was thought to be ill-defined.' Exactly what I should expect; but what is the precise meaning of the expression 'thought to be?'

mischief was apparently of two dates. There was a layer of dense stringy lymph about the chiasma, matting it tightly down, and the membranes in the neighbourhood were condensed, opaque, and adherent. Smeared all over those parts again were fresh layers of lymph; and there were marks of recent inflammation in the Sylvian fissure, and all along the base down to the medulla. The central parts were softened, and the ventricles full of fluid. I found descending neuritis in the optic nerves, and fatty degeneration in the tracts and tubercula quadrigemina. I can scarcely resist the conclusion that both these boys suffered from tubercular meningitis, limited in the first instance to a small part of the base of the brain, and that from this they both recovered under the treatment of cod-liver oil and iodide of iron, which we prescribed; but that the recovery of the boy O—— was only temporary, and was followed by relapse and death, the autopsy proving that the second attack, at least, was undoubtedly meningitis. Mr. Mann tells me that another child of the O—— family has since died of meningitis. R—— is still living. For a few additional cases the reader is referred to Nos. 20–38 in the Appendix, some of them being taken from the records of other physicians.

I could bring forward at least a dozen more cases, fully noted, in which I suspect that meningitis has existed or now exists in a mild form, my suspicion being founded upon the symptoms and supported by the ophthalmic indications. To relate them, however, would not add anything of importance to that which I have already written, and would take up much time and space. I must make a reference, however, to the appearances I have found in the eyes of idiots. I have found that a certain proportion of the idiots whose eyes I have examined—of idiots, that is, who are not congenitally idiotic, but with an idiocy due to disease—present those changes, or their results, in the optic disks which I commonly find after meningitis; while, as I have said, symptoms of mental deterioration or arrest formed a prominent feature in the after-history of several of the cases which I examined during the supposed acute stages. May not many of the idiots in our

asylums be the victims of a long-past meningitis, which has left permanent injury behind it in the brain, but which has not destroyed life? In the Appendix will be found the ophthalmic condition of a few idiots—vide table; and Talbot (No. 4) and Milner (No. 8) are good examples of the kind of evidence I mean.

I shall now notice two objections which seem to withstand the hypothesis I have proposed. The first is, that until the ophthalmoscope has been used more extensively, and its indications checked by a much greater number of autopsies than at present, we cannot say with certainty what inferences may be drawn from the neuritic or ischæmic states of the disks. It is as yet very uncertain whether optic neuritis always means cerebrospinal disease of greater or less extent, or whether it may occur as a local change alone. States of ischæmia, again, may hereafter be found to accompany states of disturbed cerebral circulation—of encephalic congestions, for example—which might give rise also to the cerebral symptoms I have described, and again subside, leaving not a trace behind. This brings me to the second and very serious objection:—That thickenings and adhesions of the meninges at the base of the brain are rarely recorded as old lesions in post-mortem descriptions. Dr. Long Fox seems to say in his paper in the fourth volume of ‘St. George’s Hospital Reports,’ that such changes are more frequent than is usually supposed. I give one of Dr. Fox’s cases in the Appendix (No. 28). It may be that our attention has not been directed to this point, and that such changes may be found more frequently if looked for more carefully; or it may be, that the membranes clear up after a long interval of time; or, again, the new formations may in time shrink into filmy or thready extensions which scarcely attract notice. Pathologists have not yet looked carefully for evidences of old meningitis; but now that I have put the question distinctly forward, I hope we shall soon have that number of able observers at work which only can settle a point of much difficulty.

I have, however, the pleasure of quoting the following opinion of Dr. Crichton Browne, whose vast opportunities of

observation at Wakefield, and whose ability and skill in the use of them, need not my poor testimony, but give great value to his statements. I may say that I made no mention whatever to Dr. Browne of my views on the matter, but simply put to him the three short questions which are given, with his replies in full.

1. Whether you think idiocy is often to be traced to tubercular meningitis of past years?

‘I have seen a few cases of idiocy which were distinctly referable to tubercular meningitis in early years, and a considerable number of cases in which I suspected a similar causation, but could obtain no satisfactory proof of it. When tubercular meningitis is so severe and well marked as to be *diagnosed*, it generally ends fatally. It is only when it is so limited, subacute, or masked as to escape recognition, that it is likely to pass into permanent forms of mental derangement or impairment, and then its previous existence can only be inferred. I am quite persuaded in my own mind that partial and subacute attacks of tubercular meningitis in infancy and childhood are much more frequent than is ordinarily suspected, and are sometimes responsible for cerebral abnormalities which the anatomists are in the habit of attributing to *synostosis et hoc genus omne*. I am satisfied that they constitute the transition stage through which precocious children pass into dulness or imbecility of intellect. A little prodigy of wit and learning loses health and weight, complains of vague feelings of sickness, giddiness, or drowsiness, and suffers from night terrors, paroxysms of rage, or hallucinations. His stomach or bowels are said to be deranged. He emerges from this *trifling* illness bereft of his superior powers, stupid, ineducable, lethargic, or perhaps a partial idiot. Tubercular meningitis has been quietly and covertly at work undermining his brain and sapping his faculties, while no one dreamt of its presence. I have somewhere published a case of moral insanity (idiocy of the moral sense and hypertrophy of the animal propensities) which was distinctly traceable to an attack of tubercular meningitis; and I have met with many

cases of perversion of character and liability to insanity which could be followed up to the same source. I have no hesitation, then, in classing tubercular meningitis as one of the causes of idiocy, although I would not certainly give it the highest, nor perhaps even a middle place, amongst these, supposing them to be arranged in their order of frequency. The causes of idiocy are very numerous and various, and I am not prepared as yet to marshal them in their proper order.'

2. Whether any of the cases of idiocy which I (the author) examined with the ophthalmoscope were likely to be such cases?

'Of the idiots whom you examined here, it seems to me highly probable that Talbot (No. 4) and Milner (No. 8) may have to date their idiocy from attacks of tubercular meningitis.'

3. Did you ever find traces of old meningitis at the base in cases of idiocy?

'My own experience in the morbid anatomy of idiocy is but limited, but I am aware that the appearances to which you refer—bands, adhesions, &c., as also thickenings of the arachnoid, and miliary tubercles of the pia mater—have been found in autopsies on idiots. I examined the body of an idiotic boy in the Warwick Asylum some years ago, and found great thickening of the arachnoid, adhesions between the hemispheres and anterior and middle lobes (at fissure of Sylvius), with fibrous bands at the base, thickening of the velum interpositum, and remarkable enlargement of the pituitary body. These changes must surely have been due to meningitis, and to this I set them down at the time. I had no history, however, to verify my impression. In another idiotic lad, whom I also examined in the Warwick Asylum, and whose case I published in the 'Edinburgh Medical Journal' for March, 1865, I noted firm adhesion of the dura mater to the skull, especially along the median line, and great thickening of it posteriorly, thickening of the arachnoid, pallor of the grey matter, and a glistening appearance and unnaturally firm consistence, entire absence of the septum lucidum, and

congestion and redness of the floor of the fourth ventricle; weight of brain, thirty-five ounces and a half.'

These notes of Dr. Browne's, forwarded to me in his entire ignorance of the object of my enquiries, are very interesting and important. The medical officers of any asylum which receives idiots might make very useful observations on this subject.

The final question is,—How constant are the evidences of the ophthalmoscope in undoubted cases of tuberculous meningitis? My opportunities of examination in such cases are not frequent; and this question could be answered far better by physicians of such institutions as the Children's Hospital in Great Ormond Street. In addition to the cases which have occurred in my own practice, however, some of the medical men in this district have also been so kind as to place cases of tubercular meningitis at my disposal for examination. I find that ophthalmoscopic examinations seldom annoy the child, except during a paroxysm of headache; and they rather please the parents, who think we are looking into the brain. I have examined the eyes in about thirty-eight cases of tubercular meningitis of undoubted characters, which were watched through their course, and ended in death. I do not include the indefinite number of cases which I have seen once or twice at the hospital or at the dispensary, and have not followed up. In twenty-nine of these cases I found ophthalmic changes; and for the most part I found them on the first examination. The changes were, most commonly, marked degrees of hyperæmia of the retinal vessels, which become clouded, swollen, dark, and tortuous⁵. These changes were often traced up to the full development of ischæmia, which appeared next in frequency. Neuro-retinitis I found in about twenty-five per cent., as it occurred distinctly in six cases. In five of the cases I

⁵ A curious change is recorded by Betz ('Memorabilien,' 7, referred to in Virchow's 'Jahresbericht' for 1869, vol. ii. p. 477). There was intense injection of the lower half of the eyeball, which appeared thirty-six hours before death. At the autopsy, a clot, 3''' long and $\frac{2}{3}$ ''' thick, was found in the right ophthalmic artery before its entry into the foramen opticum.

was present at the autopsy, and found the usual mischief at the base of the brain, in the Sylvian fissures, around the ventricles, and so on. It is somewhat remarkable that I should have met with so few exceptions to the rule of concurrence of the eye-symptoms with the encephalic mischief; but my numbers are far too small for any dogmatism. On one occasion, when I was talking the matter over with Mr. Carter, he said that he had a case of tubercular meningitis then under his care at Stroud, in which no eye-symptoms were present. He promised to examine the child's eyes again and again till death, and to secure a post-mortem if possible. He was kind enough to do so, and to place the case at my disposal. (Vide Case 30 in the Appendix.) There was no mischief at all about the anterior part of the base of the brain; but there was mischief, small in quantity and slight in degree, about the cerebellar pia mater, especially between its hemispheres. The optic nerve was not congested.

This remarkable exception tends to prove the rule we should anticipate—namely, that tubercular meningitis is only revealed to the mirror when it invades the anterior and inferior parts of the encephalon; and that the eye-symptoms, though the rule, are yet in reality accidental, and depend upon the locality of the mischief. In Mr. Hulke's cases, published in the sixth volume of the '*Ophthalmic Hospital Reports*,' part ii., I find four in which meningitis was known or supposed to coexist with optic symptoms. One of these patients recovered. In another case death followed; and in this optic neuritis was succeeding the '*Stauungs*' papilla, as in my case of Master O——, but had not, as in my case, reached the disk. Mr. Hulke points out that *Stauungs* papilla, or *ischæmia papillæ*, is not therefore pathognomonic of intracranial tumour, as has been supposed by Græfe, but here occurred as a consequence of meningitis of the base. I pointed out the same thing in my lectures '*On Optic Neuritis*,' published in the '*Medical Times and Gazette*' in 1868.

Mr. Hutchinson published in the '*Royal Ophthalmic Hospital Reports*' for December, 1866, a remarkable paper,

with tables, entitled 'A group of cases of Optic Neuritis in Children.' It is quite unaccountable to me how I managed to overlook this paper until after the publication of my remarks upon Meningitis in the 'Lancet' for May 1 and May 8, 1869. Mr. Hutchinson does not distinguish between papillary ischæmia and optic neuritis, and the latter he refers to 'arachnitis, cerebral tumours, and blood-poisoning,' meaning, I suppose, by blood-poisoning, fevers, pyæmia, syphilis, and the like.

Meningitis is, in many cases no doubt, the cause of the 'optic neuritis' in 'blood-poisoning,' as I shall prove in the three classes of cases I have instanced. Mr. Hutchinson goes on to say (p. 307), 'My attention has for some time been drawn to a group of cases in which optic neuritis occurs in children. . . . Nearly always there is a history of a severe illness, which was supposed to be fever, and was marked by delirium and other head symptoms. As the child recovered from this it was found that blindness, either partial or complete, had occurred. Frequently such children regain good health, in fact recover perfectly, excepting as regards the sight.' Mr. Hutchinson quotes also the case of 'an adult man, who had become blind after an illness attended by vomiting, pain in the head, &c.' (p. 309). In these remarks, and in the tabulated twelve cases which follow, we have the surgical attitude in the matter. These twelve patients saw Mr. Hutchinson, not because they had suffered from meningitis—indeed, it does not seem to have occurred to Mr. Hutchinson that their symptoms were due to meningitis—but because they were more or less blind. I have no doubt that all these cases were cases of recovery from meningitis, and cases in which the optic nerves had been seriously affected. In the majority of cases, perhaps, the optic nerves are much less affected, and the sign can be recognised only by the forewarned physician. Probably in the majority, or the large majority of cases of mere papillary ischæmia, the nerves may recover their health and function. I have quoted two of Mr. Hutchinson's cases in the Appendix [Nos. 35 and 36]. I have long purposed

to look for such cases in an asylum for the blind, but occasion has not favoured me.

I now ought to cite the experience of M. Bouchut, who has used the ophthalmoscope in a large number of cases of meningitis; his observations are very important, though M. Bouchut writes too enthusiastically about the ophthalmoscope to secure my willing assent to all that he advances.

M. Bouchut, in his treatise published in 1868, states that he had then examined and carefully noted the ophthalmoscopic symptoms in fifty-nine cases of meningitis, and that he had found symptomatic changes in the eye in all but two. Of these two one was insufficiently examined, and the other was of doubtful diagnosis and ended in recovery. I cannot but think, however, that these cases would lead us to overrate the frequency of neuro-retinal disturbance in meningitis. It is also stated by M. Bouchut that the eye, on the side of the main lesion in the brain, is more affected than the other, and he says that in this way the mischief may be followed from one side of the brain to the other. I do not attach much weight to this statement: the causation of the optic symptoms is very indirect, and depends on such accidents as the position of the mischief and its consequences in the venous sinuses rather than on its extent. M. Bouchut supports me in saying that although in many cases of meningitis there can be little doubt of the disease, whether we use the ophthalmoscope or not, yet that in other and more doubtful cases the early appearance of changes in the eye may be of the first importance. The same is true in the use of the stethoscope in phthisis.

In Dr. Macnamara's treatise on Diseases of the Eye, there are some very interesting observations on an injection of the optic disk which accompanies headaches induced by over-exposure to the tropical sun. He describes the disk as being intensely congested, and the 'capillaries of the retina' (sic) as being slightly hyperæmic. In fever too, brought on by over-exposure to the sun, he says the disk 'becomes bright scarlet,' and its nervous elements 'hazy and swollen, its margin being ill-defined.' Macnamara supposes that the cerebral irritation

is due to advance of the solar irritation from the retina upwards, my view would be rather the reverse; but autopsies alone can finally settle the question.

I must not conclude this section without some reference to tubercle in the choroid as symptomatic of tubercular meningitis. Dr. Moxon has been so kind as to send me an account of such a case which recently occurred in Guy's Hospital. Several such cases have been shown of late at the Societies also; as, for example, by Messrs. Soelberg Wells and Bowater Vernon, in the nineteenth volume of the 'Pathological Society's Transactions.' Tubercle in the choroid is of little value as a diagnostic sign; it has only the corroborative value that tubercle in the lung would have; and, on the other hand, it is of too rare occurrence to make its absence worth noting. As a pathological event, however, it is interesting and instructive. Its occurrence was first pointed out by Manz⁷, who published three cases of tubercle of the choroid. Cohnheim⁸ then pushed the matter forward to the extent of asserting that tubercle is to be found in the choroid in almost all cases of acute miliary tuberculosis, and he found it in the choroid of a pig which had been inoculated with tubercular matter. Cohnheim is certainly wrong in supposing that tubercle occurs so frequently in the choroid. For three years past I have examined all the cases of miliary tuberculosis in which an examination could be managed, and have not found tubercle of the choroid in a single instance. I may have examined ten cases in all. In two cases I have examined the choroid after death with negative results. In both these cases tubercle was numerous present in all three cavities. Gräfe and Leber have gone into the whole question again⁹. They give two cases in which tubercle of the choroid was diagnosed during life and found on post-mortem

⁷ Vid. 'Archiv. f. Ophth.' Bd. iv. Abth. ii. p. 120, and Bd. ix. Ab. iii. p. 133.

⁸ Virchow, 'Archiv.' May, 1867.

⁹ 'Archiv. f. Ophth.' xiv. Ab. i. p. 183; vid. also Gräfe, 'Ueber Aderhaut-tuberculose,' Berl. Klin. Wochenschr. (1867), No. 31.

examination. In the first case, a man under the care of Griesinger with tuberculosis of the pia mater, the eyes were examined by Von Gräfe, who found spots of tubercle in the choroids. After death miliary tubercle was discovered in the pia mater, and scattered through the body, and their presence in the choroids was verified.

In the second case, a child aged fifteen months, under the care of Dr. Fränkel, the eyes were examined by Dr. Leber. Eight spots of tubercle were seen in the left, and two in the right choroid. During the time of the child's illness these spots grew larger and became more evident. It is quite possible that the discovery of tubercle in the choroid might help in the diagnosis of an obscure case, but as yet, I think, it does not occur with sufficient frequency to raise it out of the domain of pathological curiosities. When it does appear, however, it has this advantage, that it reveals the morbid process to us in its kind, which cannot be said of any other of the phenomena of medical ophthalmoscopy.

The microscopical appearances of tubercle in the choroid are well described by Cohnheim (*loc. cit.*) in seven cases. He found the number of nodules very various, the smaller being transparent and the larger being caseous in the centre. They begin on the retinal surface, but when large (1 millim.), reach as far as the sclerotic surface. Cohnheim traces their origin to lymphoid cells, and not to connective tissue cells, which bears out the observations of Sanderson. In all his cases there was abundant tubercle elsewhere. Tubercle may, however, exist in the choroid when it does not exist in the meninges of the brain; this is proved by one of Steffen's cases, in which tubercle of the choroid co-existed with acute tuberculosis, but without meningitis.

Tubercle of the choroid does not generally cause loss of visual power, but I have seen a case quoted from Barozzi ('*Gaz. Med. d'Orient. Fev.*' 1869), in which tubercle of the choroid caused blindness of the left eye a month before death. There were four tubercles near the disk, and there was a detachment of the retina which no doubt was the direct cause of the loss of sight. I have not seen the original account, but

it seems that no autopsy was made. There was, however, evidence of tubercle elsewhere.

B. *Meningitis in Fevers, Erysipelas, Pyæmia, &c.*

In passing on to consider the effect of these kinds of meningitis upon the eye, I have to repeat that the nature of the disease, whether suppurative or other, has little to do with the occurrence of the eye changes,—unless it be that phlebitis and occlusion of the sinuses be more common in the suppurative forms,—but that its position is the important matter. Meningitis of the base of the encephalon, whatever be its kind, is very commonly accompanied sooner or later with disturbance of the vascularity of the back of the eye, and, in more protracted cases, descending neuritis often appears at the disk. Now, meningitis born of causes from within is very commonly basilar, if we except the chronic meningitis of drunkards we may perhaps say always basilar; while meningitis which results from an injury from without or from caries, need not be, and very commonly is not situated at the base. I say, meningitis which accompanies internal changes, though by no means always, is yet very commonly at the base, and even when a large tract and various districts are affected the base is generally included. I suppose this depends upon some greater irritability or vascularity of the membranes at this part. The meningitis which occurs in the eruptive fevers, in erysipelas and pyæmia seldom spares the base of the encephalon; and in the ophthalmoscope we have a valuable means of ascertaining whether this state be present or not. Is the violent delirium in a given patient due to meningitis, or is it only due to some functional disturbance in the encephalon and its vessels, is often an anxious question with the physician, and the help which we obtain from the ophthalmoscope is our only means of answering this question in a large number of instances. My own impression from such experience as I have is that this help is often of great value; my experience, however, is as yet too limited to

enable me to speak decidedly. I have examined the eyes of many patients in typhus and enteric fever who suffered from head symptoms, and in the majority I found nothing; in three fatal cases, however, I saw ophthalmoscopic evidence of the meningitis which was found after death to have been present. In five more cases I saw evidence of meningitis, as I suppose, which ended in recovery of health, but in partial or complete loss of sight; and in two cases I found excessive vascularity at the back of the eye, which subsided during convalescence. That loss of sight occasionally follows the continued fevers has long been noticed by clinicians, and is, I believe, in all cases due to meningitis¹⁰. [Vide Appendix, Nos. 40-46.]

Stellwag von Carion gives a remarkable case in which he saw optic neuritis in the eyes of a patient in typhoid fever. Some years later this patient died of some other disease when old organized exudations were found in the membranes at the base of the brain. This is a most remarkable case, and cases of the kind might often, no doubt, be discovered in our asylums.

Von Carion also says ('Dis. of Eye,' Trans. Lond. 1868, p. 672), that in rare cases the optic atrophy does not appear until long after the disappearance of the meningitis, and that it is then due to cicatricial shrinking and obliteration of the portions of the membranes which have been inflamed. He adds that in such cases the autopsy here shows actual tying together of the nervous cords by cicatricial tissues. This seems to me one of those plausible descriptions which may turn out to be purely imaginary. I have, however, a very remarkable case under my charge at present. A young woman, Barbara H——, now aged 14, had 'fever' when three years old. She seemed to get well at the time, but has been liable to attacks of vomiting ever since. During some years they recurred once a fortnight,

¹⁰ Cases are frequently described of optic atrophy, with or without some neuritis, as following diphtheria, &c., and due to the 'dyscrasia.' Such explanations, especially if without autopsies, as is commonly the case, are too subtle for my handling.

but during last three years every week. The attacks last two or three days, and are preceded by drooping of the left eyelid for twenty-four hours antecedently. In both disks there is subacute neuritis, due to long-continued 'Stauungs-papillæ.' She is not conscious of loss of sight, but on testing she cannot read small pica easily with the left eye.

I have notes of the optic nerves in three cases of *pyæmia*. In two of these cases, which occurred in private practice, I examined the eyes during life, and found in one that the appearances were normal, while in the other there was a well-marked ischæmia in both disks. I heard nothing more of either case. The third case occurred in the Wakefield Asylum [Appendix, No. 45]. I did not examine the eyes during life, but Dr. Browne was so good as to investigate the state of the optic nerves for me after death. He found limited meningitis with some exudation about the chiasma and a highly injected state of both optic trunks. It is probable that in this case there would have been visible changes in the vascularity of the disks during life. The patient died delirious, but with no marked symptoms of meningitis. I am sorry that I had not the nerves for microscopic examination. The occurrence of optic neuritis in connection with *pyæmia* is mentioned by several writers, but no doubt in many of these cases the change was rather ischæmic than neuritic.

I hope the use of the mirror will not be omitted by those in charge of our fever hospitals. The permanent amaurosis which is often said to have followed measles, scarlet fever, small-pox, &c., is probably of like causation, and the mirror may give us important information concerning any severe delirium, or other head symptoms occurring in consequence of these fevers. I have but few notes of personal observation in such cases. Cerebrospinal meningitis is a fever which I have never seen; in it extensive mischief often appears in the eye, and mischief which would make it impossible to see the fundus. 'Optic neuritis' has, however, been noted in several cases; as, for example, by Schirmer, 'Klin. Monatsbl. f. Augenh.,'

1865, p. 275; and Mr. Wilson says ('Dublin Quarterly Journal,' May 1867), that 'amaurosis is not an uncommon sequence of cerebrospinal meningitis.' It seems likely that the mischief commences in the inner coats; but I am disposed to suspect that choroiditis rather than neuro-retinitis is the first disorder. I am disposed to be of this opinion because a curious state of the inner coats of the eye has been described in puerperal fever, which is worth an allusion in this place, though it partakes rather of the nature of general ophthalmitis. It consists of a purulent choroiditis, and is interesting, inasmuch as it may throw more light upon the process of capillary embolism so well described by Bastian and others, and also as it seems to lead on to that more general destruction of the eye which is seen in epidemic meningitis. I know nothing of this puerperal choroiditis of my own knowledge, and am entirely indebted for my information to a paper by Knapp in the '*Arch. f. Ophth.*' xiii. 1. The paper is a long one, and well illustrated. Knapp describes the parts in three cases. The purulent inflammation begins in the chorio-capillaris, and is apparently due to capillary infarctions which soften and break up in pyoid forms. From this layer the inflammation extends to the stroma of the choroid, which proliferates and runs down into fat and pus, and thence the retina, the pars ciliaris, the vitreous humour, and even the capsule of the lens are invaded. The process seems to recede before perforation takes place. If this plugging and purulent change be traced out in the chorio-capillaris, it will be a fact of great interest and importance in connection with the hypothesis which attributes a like origin to the other local formations of pus in pyæmia.

This is, perhaps, the place in which to discuss the peculiar and singularly interesting changes observed in the fundus in cases of cholera, by Von Gräfe¹¹. After describing the well-known sunken, upturned, bloodshot, and dry eye-ball, he says of the changes seen with the mirror, that, except in the final agony, the circulation in the retinal vessels is always to be

¹¹ '*Ophthal. Beobacht. bei Cholera,*' *Arch. f. Ophth.* vol. xii. part ii. p. 198.

made out, although the branches of the central artery are extremely thin, as may be seen also in other states of general disorder. But in striking contrast to the pale and half-filled arteries are, in the stages of asphyxia at least, the very dark, even blue-red veins which are visible up to their finest ramifications, without, however, being very full or very tortuous. Not infrequently the aspect of the disk is also changed, and instead of the uniform rosy tint of health, it appears of a pale lilac. There does not seem to be any loss of visual power. These very interesting observations are one more proof of the value which the eye mirror may be to us in ascertaining the kind of changes which the circulation of the brain undergoes in asphyxia and in various other kinds of general disturbance. Finally, I must warn the reader against the supposition that the transient debility of vision, which often follows or accompanies severe constitutional disease, signifies meningitis, or indeed any other organic disorder, unless it be a remediable anæmia with or without œdema in the optic nerves and base of the encephalon. The disk may be white and the vessels small in these states, or there may be some opalescence of the fundus; the field of vision, however, such as it is, remains complete, and the functional deficiency passes away in a few days or weeks under tonic and nutrient treatment. Paresis of accommodation often accompanies this kind of amblyopia; and Dr. Hughlings Jackson makes the interesting remark that diphtherial paralysis differs from all other kinds of paralysis with which he is acquainted in seeming to have a great preference for those branches of nerves which pass through the ganglia of the sympathetic. For instance, in the so-called diphtheritic amaurosis the whole of the third nerve is not paralysed, but only those branches which pass through the lenticular ganglion. The facial is not paralysed, but those branches of nerves given off by Meckel's ganglion to the palate. Next, as to the otic ganglion. Hearing is practically not affected at all in the general run of cases, as the accessory muscles of the ear have far less to do with the functions of the organ of hearing than the ciliary muscle has with sight. But in one

case, that of a well-educated medical man, notes of which had been furnished to Dr. Hughlings Jackson, there was slight defect of hearing. It was not enough to render the patient unable to converse, but, to use the patient's expression, it 'rendered music unintelligible.' Next, there was in this case too, defect, not loss, of taste, due probably to an affection of the branch of the facial (the chorda tympani), given off to the submaxillary ganglion.

C. Syphilitic and Rheumatic Meningitis.

These two forms of meningitis, like the tubercular, are proliferative and affect chiefly the base. One of them is so common that I need not here spend time and space upon it, while the other will receive as scant measure for the opposite reason, namely, that it is almost unknown. Of rheumatic meningitis there appear to be two kinds; the one kind resembles, both in character and time of appearance, the other serous inflammations which attack in the course of rheumatic fever, while the other is a very chronic process attended with thickening and adhesion. My information concerning these states is almost entirely derived from the treatise of Gintrac¹², for I have no experience of either of them. I have seen four cases of so-called 'cerebral rheumatism' with very high temperature; one of these recovered, and in the three which died no meningitis was found, but only the congestion usual in such cases. Probably many cases are reported as meningitis in which no such process existed. I examined two of the cases alluded to with the mirror, but saw nothing abnormal unless it were some fulness of the veins. The mirror may have the power of distinguishing between the presence and absence of meningitis in these terrible cases. Of chronic meningitis following rheumatic fever, I have seen but one case, and in this I have not the control of an autopsy. It occurred in a man named Abraham Waddington, æt. 35, who was admitted into the Wakefield Asylum for violent mania, which was

¹² 'De la Meningite Rheumatismale,' Bordeaux, 1865.

a sequence of rheumatic fever. From the symptoms which continued during a long period of subsequent observation, Dr. Crichton Browne believed that chronic meningitis was present. When I saw him he had much improved, and was, for the time at least, in a very manageable condition. With the right eye he could read No. 6 (Jäger) with difficulty, with the left eye No. 3 with difficulty. Both disks were distinctly though not extremely atrophied, the right being worse than the left¹³.

My experience of syphilitic meningitis, on the contrary, has been considerable, and I can appeal to much experience embodied in many careful essays from that of Thomas Reid to the present moment. It is unnecessary for me therefore to add to this volume any more cases or much explanation in this place; but I may refer the reader to my paper on 'Syphilitic Disease of the Nervous System,' in the fourth volume of 'St. George's Hospital Reports.' Active proliferation is very significant of this affection; though partial it often affects the base, and especially that part of it which lies on the basilar process, so that the nerves of the face are very frequently involved. True neuro-retinitis propagated downwards to the eye is a very common accompaniment of this disease, the connection being more certain than in any other meningitis. Ischæmia of the disks is less common in this form, but atrophy of a primary kind is not uncommon¹⁴, and depends upon an intracranial severance of the optic nerve due to strangling. It is very important to examine the disks in all cases of suspicious headache; for in such I have often found neuro-retinitis, a discovery which of course is important in many ways. I will only add that syphilitic neuro-retinitis is more

¹³ There are certain palsies of the orbital muscles which are recognised as rheumatic by ophthalmic surgeons. These palsies do not form part of the present chapter, and if they be mere muscular conditions they will not form any part of the subject. If, however, they are found to consist in any visible disturbance of the nutrition of the nerves, they may throw light upon many points of interest to the student of nervous affections.

¹⁴ I found it, for instance, in a case the details of which, with the autopsy, were published by Mr. Lawson Tait in the 'Medical Times' for Feb. 1869.

amenable to treatment than other kinds, and may sometimes be cured by anti-syphilitic remedies and atrophy thus averted. Syphilitic neuro-retinitis is, however, nearly always well pronounced and, as it is common, no cases make better specimens for students or beginners in ophthalmoscopy. Syphilitic retinitis is described in a subsequent chapter of this volume.

D. *Meningitis of Drunkards.*

This is a very chronic change ending in thickening and opacity of the membranes, especially over the convex surface of the encephalon. It is familiar to the medical officers of asylums, and it is there that I have met with it. It seems to affect the base much less than the convexity, and I do not know that it has any effect upon the optic nerves. These are often degenerated in drunkards, and the vessels injected, but these effects do not seem to be due to any meningitic process.

E. *Traumatic Meningitis.*

Traumatic meningitis, like the pyæmic, is commonly suppurative, but it differs from the pyæmic in having little preference for the base. The mirror is seldom of use in these cases; they rarely present much difficulty of diagnosis, and unless the mischief be at the base, the meningitis around it will not interfere with the optic nerves. Sometimes, indeed, this inflammation spreads away from its point of origin, and if it spreads over the encephalon it may well affect the base also; but any optic signs in such a state of things would have but little importance beyond that of mere curiosities. In some cases of traumatic meningitis, indeed, I have found the mirror of some use, and that is in cases of caries of the petrous bone resulting from disease of the ear. The occurrence of marked changes in the disks in such cases is a very serious event, but we have no means of distinguishing whether they are due to abscess, to meningitis, or to phlebitis and thrombosis of the sinuses. I make a practice, however, of examining

the eyes in all cases of suppurating ear. Vide Appendix, Nos. 42, 43. For some further remarks on this subject I may refer the reader to my sections on Encephalic Abscess and on Fracture; also to Dusch's monograph on Thrombosis of the Cerebral Sinuses, published by the Sydenham Society.

VI. CONCUSSION AND FRACTURE.

I have no experience of the optic nerve in cases of recent concussion save this, that I have examined some half dozen cases with negative results. In these cases, however, I had no reason to expect any visible changes, as the only defects of the special sense seemed to be those of indefiniteness or illusion, which might well depend merely upon changes in the encephalic centres. Cases, however, do occur, and occur frequently, in which there may be some more decided interference with the perceptive power of the retina, or with the conducting power of the nerve. Mr. Le Gros Clark, in his Lectures on surgical diagnosis, gives a very remarkable example of this kind. He says (p. 52): 'A youth who had fallen on his head was admitted, with slight concussion, into the hospital. His most marked symptom was loss of vision; he could not even perceive light. This condition was very transient; in half an hour he could just detect the presence of my fingers when interposed between his eyes and the bright daylight; and in the course of another hour he had recovered natural vision. He made a speedy recovery.' Mr. Clark is disposed to refer the blindness rather to the encephalic centre than to the retina. However this may be, we have here and in like instances good examples of the remarkable occurrence of functional arrest in nervous parts which are supposed not to be structurally degraded, but to have assumed perhaps some isomeric state. That the optic nerve in such states is not obviously injured, it is the interesting part of the ophthalmoscope to discover; and we have here one more instance of the important fact that changes of a congestive, inflammatory, or atrophic kind in the disks must have some time for their development.

If we leave cases of recent concussion, however, I come upon so large a number of cases, both in my own practice and that of others, that I have no lack of materials, whatever use I may make of them.

When I omit all reference to railway accidents, which are full of pitfalls for the enquirer, and take only cases where there is no likelihood of deception, I still find a large number in which amaurosis has followed a blow upon the head. In passing, let me say, first of all, that in a very formidable number of cases of syphilitic intracranial disease, and of tumour, we have a history of a blow. Like other observers, I had long been in the habit of disregarding the blow, as we know but too well how natural it is in seeking for a cause to fix upon the most conspicuous antecedent; and this is especially true of the class of patients who frequent hospitals. Not in hospital practice alone, however, but in private practice also, and in circumstances where I have been able to weigh and compare statements, I still find that we have histories of blows on the head at spots where a tumour or syphilitic mischief is subsequently found. In three several cases have I found a glioma, a hard sarcoma, and a vascular sarcoma respectively within skulls which had undoubtedly been hit hard on the region above these neoplasms. So many of my patients with intracranial syphilis have complained of blows as the cause to be blamed for the event, that at one time I began to wonder whether concussion of the base was the cause which determined syphilitic pachymeningitis to this region. I shall be glad if any of my readers who have observed that a syphilitic outbreak within a skull followed a blow upon it, or that the growth of a neoplasm in like manner followed such a blow, will tell me of the case¹⁵.

The remote events, however, which usually follow severe concussions, with or without fracture, are four—hæmorrhage, abscess, meningitis, and sclerosis. The two latter events

¹⁵ While correcting this proof I happened to meet Dr. Hughlings Jackson, and this question came up. He was much in favour of my supposition, that blows do in many cases determine an outbreak of intracranial syphilis. I think a like causation may hold in syphilis of the shins.

favour the occurrence of amaurosis, and we may sometimes distinguish between them with the mirror. I give several cases of secondary meningitis in the Appendix (Nos. 53, 54, 56), and I may refer especially to the cases recorded by Mr. Hutchinson and Dr. Slack (Nos. 53 and 64). Sclerosis of the brain mass below the seat of a blow has been found in many cases, and the hardened portion is sometimes called a tumour. Cases Nos. 58 and 59 in the Appendix, which I quote from Mackenzie, and Case 60 from my own notes, are good instances of this kind of change, with its ultimate effects upon the optic nerves. I fancy this may be a commoner cause of amaurosis than meningitis, especially if atrophy be the first stage in the change, and I must refer to the section on Sclerosis for further explanation of the mode of it. While meningitis will often produce neuritic symptoms, sclerosis will always produce primary atrophy. The effects of abscess in causing optic changes are also discussed elsewhere, as are the effects of hæmorrhage. I have said that recent hæmorrhage has no power over the optic disks; and if an old hæmorrhage affects them, it is probably by intermediation of sclerosis, so that abscess or meningitis may be paired together as links between blows on the head and amaurosis, while hæmorrhage retires behind sclerosis.

In cases of *fracture* we find, if we direct our attention to the bones only, that affection of sight is a rare consequence, though many instances of this kind of interference are recorded. When fracture sets up secondary meningitis about the base, the consequences may well be seen in the disks, but such signs would form too small a part of the assembly of symptoms to be worth much in diagnosis. For my own part I have examined about a dozen cases of fracture, but if a slight dimness of outline be excepted, I have never discovered any important changes in the fundus. If such changes do occur, not as a consequence of meningitis, but as a direct result of the fracture, the nerve is either compressed by a displacement, or the same kind of ecchymosis occurs in the retina or nerve which is commonly seen in the conjunctiva,

and for the same reasons. Examples of all these results are given in the Appendix (Nos. 61, 62, 63, 64). In one case of fracture which I examined with the mirror, the left eye-ball was full of blood. Amaurosis as a direct result of displacement of bone, or of very limited meningitis, is more likely to be unilateral than when it results from other changes, except in the one case of aneurism of one anterior cerebral artery. I suppose extensive hæmorrhage at the base, or indeed anywhere upon the encephalon, might cause neuritis or atrophy of the disks if it took on the cystic change and became an intracranial tumour.

VII. *HYDROCEPHALUS.*

Hydrocephalus, or simple dropsy of the brain, is very destructive to the optic nerves, and blindness is often due to this cause. Ischæmia papillæ seems in many cases to be the earliest change, as I have certainly noticed it in early cases more than once. Herein I am obliged to differ from Gräfe, who has only found simple white atrophy in cases of hydrocephalic amaurosis. It is likely that Gräfe, however, does not see hydrocephalic children until the mischief is old and the loss of sight obvious. If the affection be at all extreme, the disks and retinas become wholly disorganised, and the optic nerves become atrophied from pressure, or from abolition of their function. With the mirror we see the disks atrophied, their outlines blurred or lost, the vessels distorted or closed, and brownish or whitish patches, blotches, and streaks upon the retina; some of these being old hæmorrhages, others exudations and fatty degenerations. I believe these changes are mainly due to pressure, or severance of central connections, though it would be difficult to deny that some subinflammatory action and proliferation is often mixed up with the death by over-distension and prevention of nutrition. A dropsical effusion pressing down the floor of the third ventricle will, of course, tend to press upon the chiasma. M. Bouchut says that with the mirror we may distinguish the large square heads of rickets from hydrocephalus. He would say, then, hydrocephalus had occurred in the course of rickets in the

following case of a little child formerly under my care at the infirmary¹⁶. She was about three years old and the youngest of the family. There was a distinct history of rachitic symptoms, enlargement of the ends of the long bones, and some abnormal curvature in their length. The chest was characteristic also. The face was square and the head large, but neither its size nor the state of the fontanelles was sufficient to enable us to diagnose hydrocephalus. With the mirror the disks appeared swollen and vascular, and the retinal veins distended. I have lately admitted another little child of weakly constitution into the infirmary, whose eyes I examined simply to show one of my pupils how to examine the eyes of children. To my surprise I found atrophy of the optic disks to the second degree. On enquiry from the mother I learned that the child, which was nearly a year old, had not begun to 'take notice' as she had hoped, and I believe it was half blind. I watched the child carefully. In a fortnight afterwards its head began to enlarge, and in three weeks more had attained a considerable size. The child is now distinctly hydrocephalic, and is not improving under treatment. There have been no symptoms of meningitis. Here we probably had simple atrophy as the first optic change, and in every point of view the case is one of great interest. It teaches us, among other things, that the eyes of all children which 'take notice' late ought to be examined with the mirror.

Tumour or other encephalic diseases often act upon the eye by the mediation of hydrocephalus. A case was lately recorded in the '*Lancet*' (March 21, 1868), in which there was cerebral dropsy due to the pressure of a cyst upon the venæ Galeni. The child is reported as 'having the special senses, sight, &c., perfect.' The case passed out of notice, and when seen again blindness is reported. No doubt changes in and near the disk might have been seen long before much disturbance of vision was noticed. I must warn the reader against accepting any cases on the negative side in which the mirror was

¹⁶ Dr. Dickenson, in the '*Lancet*,' July 16, 1870, says that of twenty-six hydrocephalic children which had come under his care, signs of rickets were noted in nineteen.

not carefully and repeatedly used. When, on the use of the mirror, we find *ischæmia papillæ*, we should infer that the pressure is upon the venous system; when we find atrophy, we should infer that the optic changes are rather due to pressure within the ventricles upon the thalami, and through the *crura cerebri* upon the tracts, or downwards upon the chiasma. There seems to be no doubt that tumours of the posterior part of the cranium, such as tumours of the vermiform process of the cerebellum, do act upon the optic disks by the intermediation of hydrocephalus, which is due itself to pressure upon the veins of Galen, or upon the lateral sinuses, or perhaps to closure of the communication between the fourth ventricle and the subarachnoid space described by Mr. Hilton¹⁷. I cannot say whether in cases of suspected hydrocephalus, as, for instance, in the large head of rickets, the absence of ophthalmoscopic signs has any or much value in diagnosis. A good deal of experience is necessary to determine such delicate points as these; I am disposed, however, to think that the absence of such signs should claim a place among other considerations.

VIII. INTRACRANIAL TUMOURS.

It is with much hesitation that I enter upon the section of encephalic tumours, for this is the classical ground of medical ophthalmoscopy. It was to the diagnosis of encephalic tumour that Von Gräfe first applied the mirror, and he based upon such observations his well-known introductory paper in the seventh volume of his 'Archives' (for 1860), 'On the Complication of Optic Neuritis with Diseases of the Brain.' Since that time cases of tumour have formed the staple of similar observations made by himself and many others up to the present time. I had fair grounds for hoping therefore that with such materials I should find myself in a position to sum up more or less conclusively what has been said on this part of the subject, if not to formulate with something like completeness a series of

¹⁷ Dr. Dickenson has again demonstrated this opening, and proved that it is traversed by a fluid so thin as the cerebrospinal fluid.

definite propositions. It is with a sense of disappointment however that I find myself, at the end of much reading and of much clinical observation, very far from any such results. I am not in a position even to indicate, with anything like finality, the actual value of the presence or absence of optic change in the diagnosis or exclusion of encephalic tumour; nor am I able to say with anything like certainty what are the intermediate processes which connect these changes in the head with inflammatory or congestive changes in the disks. I must content myself then with the humbler task of revising and setting forth the facts as they seem to have been made out by myself and others, and must still look forward to the time when these facts shall be comprehended in some more general laws.

Encephalic tumours are not among the common things of practice, even in the practice of those who, like myself, are connected with large hospitals. Still less commonly can we trace these cases from their clinical beginnings to their pathological ends. The cases are tedious, they go from place to place, and from doctor to doctor, and at last die when we are least expecting it, or are least able to secure an autopsy. The number of cases of encephalic tumour which have passed before me must no doubt be considerable, and these have in many instances been well watched for a time, but I cannot even now count more than eight cases which I have watched from first to last in life, and have examined after death, and eight cases is but a small number to reason upon. I fear it is a deceptive process to make up cases by tacking fragments together, but it seems that if I wait for more complete ones I may postpone publication indefinitely. In like manner I might postpone this work even longer did I wait to attain to a clear perception in my own mind of the nature of many of those processes in nervous pathology, which nevertheless bear very closely upon the interpretation of changes secondary to encephalic tumour.

Another difficulty there is in this, that the great majority of published cases of tumour are useless for our present purpose. With the beaver-like instinct of an animal collecting

materials for a book, I had made a catalogue of a vast number of cases, both from home and foreign journals and from treatises on encephalic diseases. Taking Ladame's categories as a basis, I had added so many new cases that a very formidable list was obtained. But when I came to analyse this list, which had cost so much trouble, and from which I hoped to obtain at least some rough generalizations, I was obliged to admit that it was almost useless. I became convinced that it was impossible to give any importance whatever to those cases in which no mention was made of loss or disturbance of sight, nor could I attach much value to the majority of those in which vision was actually declared to be unaffected. If the reader has followed me to this point, I have, I hope, convinced him that even a careful testing of vision can have no value whatever in the opinion of any observer who knows how marked the changes may be in the fundus of an eye which nevertheless can see well, or can see well enough to satisfy a patient struck down with mortal disease. Changes in fact of a congestive kind may no doubt be present throughout the duration of the malady without giving rise to any visual disturbance whatever, and neuro-retinitis has been recorded in cases of tumour where vision was never in the least degree affected from first to last. (Vide e.g. Appendix, No. 91, &c.) Nay, more, in those cases of encephalic tumour in which the mirror has actually been used, and its revelations recorded, we find many recorded in a very unsatisfactory way, or more commonly we find that the mirror was used once only in a case which lasted perhaps one or two years. To say from such an observation that the optic nerve was not implicated, is of as much value as to record the fact that the patient's limbs were examined on a certain morning and were found to be unpalsied. Though I do not regret that I have made the list, I feel however that for these reasons I must not add to the cost and size of this little book by publishing it. On the other hand, if the general question be asked,—Is the ophthalmoscope of any use in the diagnosis of tumours?—this question must be answered decidedly and emphatically in the affirmative. Although we

cannot yet say exactly when to expect eye symptoms, what conclusions to draw from them when present, or finally, how to explain their occurrence in many cases, yet I hope to show that some information of a very important kind is to be had in this way. When a case comes before us in which encephalic tumour may be present, the following questions among others present themselves for reply :—

1. Is the case one of encephalic tumour at all ?
2. If so, what is the nature of that tumour ?
3. Of what size is it ?
4. Where is it situated ?

We may soon reduce these four questions to two, for the purposes of our present enquiry. With the second question we have not indeed to deal, for the ophthalmoscope is a clinical rather than a pathological instrument. It has certainly been made an objection to the value of ophthalmoscopic researches that the changes in the eye, if present, can tell us nothing of the nature of the intracranial tumour. Of course I admit that the discovery of cancer or of tubercle in the eye is of too rare occurrence to give any meaning either to their presence or their absence. But what does hemiplegia tell us of the nature of cerebral tumours ? Nay, what does the stethoscope tell us of the nature of pulmonary changes ? It gives us only an evidence of changed physical conditions, and every physician knows how misleading this evidence may be, if it is not weighed with all the other circumstances of each case.

The third and fourth questions are really one, for the tumour is situated in every part to which it extends. We have then to enquire, first, whether any given case be one of tumour or not, and if so, then what parts the tumour occupies at any given time. Under the name of tumour it is convenient to include also cysts, aneurisms, exostoses, collections under the dura mater, &c., as well as sarcomatous, gliomatous, tuberculous, carcinomatous growths, and the like. A tumour will then be for us any enlargement or thickening encroaching upon the intracranial chamber.

The frequent occurrence of dim vision or of blindness in connection with encephalic tumour has long been known, and

before the eye-mirror came into use, the disturbance of vision was counted as a common symptom. Friedreich¹⁸ found disturbance of sight to occur in twenty-six out of his forty-four cases, and Ladame, who tabulates 331 cases, estimates the disturbance of vision to occur in about one-half of the whole number. These estimates are founded of course upon the rough evidence of the patients themselves. In many cases, no doubt, slight visual defects have been overlooked or regarded as unimportant or uncertain, and undoubtedly in many more cases the eye-mirror would have revealed changes at the back of the eye which no other means could have detected. Dr. Hughlings Jackson, whose cases of encephalic tumour are controlled by skilful ophthalmoscopic examination, says, 'Tumour is nearly always attended by optic neuritis.' ('Medical Times and Gazette,' Aug. 15, 1868.) My own opinion certainly is, that changes either of a congestive, neuritic, or atrophic character may be found in the disks at some time or other in the course of almost all cases of intracranial tumour. The diagnosis of a case of this kind is therefore incomplete unless the eye-mirror has been carefully and repeatedly used. It being thus conceded that changes in the optic nerve are a frequent, though not perhaps a constant, accompaniment of intracranial tumour, we have to enquire, in the next place, what are the conditions of this coexistence, in order that we may know, if possible, when to expect it, and what conclusions to draw from it when present. We shall see presently that the situation of the growth has much to do with the appearance of the changes in the disks, and also with the time of their appearance. But we must be satisfied, if possible, as to the mode of the initiation of the papillary congestion, optic neuritis, or atrophy which we may discover before we can hope to obtain a knowledge of the processes by which they are established. I had thought that the way was clear to the establishment of the intermediate changes, and in the former parts of this book I have laid down certain inferences concerning their nature which I supposed to be

¹⁸ Friedreich, Beiträge z. Lehre v. d. 'Geschwülsten innerhalb der Schädelhöhle.' Würzburg, 1853.

unquestionable. Since those parts were written, however, the attribution of changes in the nutrition of the optic disk to pressure, or to the travelling downwards of irritative action, has been seriously disputed. I have not re-written my earlier chapters, because I still hold the views therein expressed, but the importance of the counter hypothesis, and the eminence of its supporters, are such that I take occasion in this place to reconsider the causation of neuritis and atrophy. Regarding pressure and travelling irritation as insufficient to explain the phenomena of optic neuritis and atrophy, another hypothesis has been substituted for these which is said to have greater explaining power.

I do not know who is to be called the author of this newer hypothesis, but I find it clearly laid down by Benedikt¹⁹ in 1868, and I believe that it has been adopted in England by Dr. Hughlings Jackson. I cannot therefore dismiss it without very careful discussion, and the question is worth discussion, for it bears not only upon optic neuritis, but upon the interpretation of many other secondary changes in the nervous system.

I shall set forth the new hypothesis in Benedikt's own words, and shall then proceed to test its strength. After urging, as Dr. Jackson and myself have so constantly done, that every case of disease of the nervous system must be watched with the eye-mirror, 'for very often a high degree of neuro-retinitis will continue for a long time without any disturbance of vision whatever,' and after referring to the curious constancy of the bilateral change, and that the changes occur in connection with mischief which is far away from the nerves themselves, as, for instance, in the case of tumours of the pons and of the cerebellum, he proceeds as follows, in a passage which I think it well to translate word for word, as the original may not be easily accessible :—

'Ophthalmologists have long known these facts, though in connection with another set of ideas. The conception has gained ground everywhere that the trophic disturbances are due to disturbances of the circulation set up by direct or indirect

¹⁹ 'Elektrotherapie,' Wien, 1868, p. 249 and seq.

pressure (α). This view can only be directly proved in a few cases, and in many cases it has no precise significance. If, for example, as was the fact in one of my cases [recorded in the Appendix, No. 93], a tubercle grows on the base from the lower commissural layer of the pons, whereby the pyramidal strands are pushed so gently aside that no trace of any signs of pressure are set up around, and, nevertheless, neuro-retinitis is present, there is then scarcely any sense in which we can pretend that pressure has been exerted upon the vessels of the eye. Still less probable is this mechanical theory in the case of small tumours of the cerebellum. Besides, there are other things also against this theory. Therapeutic experience has taught us that such neuro-retinitis can be entirely reduced without any diminution in the size of the tumour (β). Moreover, the neuro-retinitis, as we know from clinical observation, very often sets in stormily with symptoms of irritation, especially with headache (γ). And it is hard to see why the congestive phenomena either primarily, or indirectly by means of exudation into the brain, should set up mechanical hindrance in no other circulation, and should set up pressure upon no other nerve district than that only of the eye (δ). Thus the mechanical theory suffices for certain cases only (ϵ).

‘There is the further notion, that in cerebral affections the neuro-retinitis is due to a neuritis descendens. This view explains certain cases only. Were the neuro-retinitis due as a rule to the propagation of inflammation downwards, then the disturbance in function ought frequently to come on without any pathological appearances in the disks. The pathological process at any single part of the nerve bundle ought as a rule to produce disturbance of function before the process has reached the periphery, and this the more, as it must be more active at its starting-point than at a part secondarily affected. A neuritis descendens can then only be admitted if the amblyopia sets in without ophthalmoscopic appearances, and the changes in the disks show themselves by little and little (ζ). Moreover, on this view all cases would be unexplained in which the progress of the inflammation on to the course of the optic nerves cannot be proved, in which, that is, the original affec-

tion does not lie in their course (η). In the third place, the neuro-retinitis and the functional disturbance often come on quite suddenly, and frequently like a shock, which most certainly cannot be reconciled with an anatomical process propagated by continuity (θ). Fourthly, in the majority of cases the original process is neither an inflammation, nor does it set up inflammation in its neighbourhood; and if we are not to interpret the neuro-retinitis as inflammation in the old sense, but as hyperæmia, with swelling and subsequent growth of the interstitial tissue, yet if neuro-retinitis is understood to be a propagated anatomical process, this process must be demonstrated in complete continuity from the immediate neighbourhood of the original affection up to the disks, which in many cases at least is not possible (ι). Secondary atrophy, both ascending and descending, appears in many cases, and we do not accurately know the conditions under which it occurs; but, for the above-named reasons, the neuro-retinitis, in the greater number of cerebral and cerebellar affections, cannot be regarded as neuritis descendens.

‘Now in order to know how cerebral processes, whether they encroach upon the course of the optic fibres or not, or whether they are bilateral or not, influence the optic nerve by trophic changes, we must consider those processes in general which in local diseases of the brain take place in its other parts; and, above all, we must enquire whether slow-growing local diseases in the brain are to be regarded as local trophic processes, as on the answering of this question hangs the proper understanding of most cases of neuro-retinitis symptomatica. We see, for example, in the development of cerebral tumours, that at certain periods, periods which apparently correspond to those of more rapid growth, widespread symptoms appear; headaches before all, which are variously localised, and may attain a degree of severity which deprives the patient of consciousness; then dizziness, sundry pareses and palsies, singing of the ears, and finally, amblyopia and amaurosis. In other cases the symptoms advance less boisterously, but are more abiding. These symptoms may then subside again for longer periods and the patients seem well.

What is the anatomical process which calls forth these phenomena in remote parts of the brain? Apparently not direct irritation of the tumour upon its own environs, for a number of these symptoms are wholly independent of the site of the disease. The ophthalmoscope teaches us rather that these symptoms depend upon hyperæmia with swelling (κ).

‘On the other hand, we frequently see—especially in atheroma of the cerebral arteries—such vascular storms (*Gefassstürme*) arise, which lead to the sudden onset of unconsciousness, hemiplegia, paraplegia, &c., and quickly pass off again, and a few autopsies made soon after point out that attacks of this kind need no permanent pathological process for their occurrence, but that hyperæmia with swelling suffices to induce them, and that these vascular storms depend at one time upon hyperæmia alone, at another upon hæmorrhage also, &c. (λ).

‘That in acute local diseases, as in cerebritis, the circulatory changes are still less local in distribution, scarcely needs to be explained.

‘Thus we see that localised intracranial diseases set in with vasomotor disturbances which affect the whole or a great part and remote districts of the brain, and, among other things, neuro-retinitis appears as a sign of this vasomotor excitement.

‘No one will look upon this widespread vasomotor disturbance as a diffuse irritation of particular vessels; in local affections within the skull we have rather to deal with coincident or preceding neuroses of the sympathetic fibres, with a local fever, as it were. Symptomatic neuro-retinitis thus, in most cases, depends upon a morbid innervation of the sympathetic, which again is a symptom of manifold cerebral processes.

‘We now thoroughly understand how it is that we find a morbid sensitiveness of the sympathetic in most cases of brain affection which have once become chronic.

‘It is now come nearer to our understanding also how it is that with tumour of the pons an affection of the cortical substance may come on which leads to dementia; further, how the most various affections of the brain in all sorts of places produce pain in the districts of sensory nerves, how suddenly

palsy of the respiration or of the circulation sets in, how hydrocephalus also is established in those cases where there can be no notion of a creeping of the process onward by continuity to the lining of the ventricles.

‘All these processes depend upon sympathetic vasomotor disturbances, which in certain particular places, for instance, in the lining of the ventricles and in the optic nerves, have a special tendency to become chronic and to beget secondary trophic disturbances (μ). In the case of the disks this special disposition is explained by the passage of the vessels of the retina through the rigid fibrous capsule of the sclerotic. If now a strong and active hyperæmia with swellings sets in, the ebb of the blood is prevented, and stoppage occurs, which is at first manifest in dilatation of vessels and growth of connective tissue, and subsequently in the atrophy of the vessels and of the nerve tissue consequent upon the swelling of the connective tissue.’

Benedikt then goes on to argue from his own clinical experience, that the control of the optic nerves by the sympathetic is proved by the therapeutic success which follows the galvanisation of the sympathetic in neuro-retinitis; into this part of his argument I cannot now enter. I have translated these pages word for word, because the views here urged by this able physician are not only very important to the matter in hand, but have bearings both clinical and pathological of the widest interest. In order to discuss them with the care they require, I have lettered the important points, so that I may omit none, and may make references without repetitions.

Benedikt has not, in the first place, quite comprehended the meaning of those observers who attribute the papillary changes to intracranial pressure. Although pressure directly or indirectly applied in the course of the ebb of the circulation (α) will set up these changes, yet the agent in many cases is not mere local pressure, but a general elevation of intracranial tension due to an excess of its contents. The exquisitely packed and mobile contents of the cranium are almost as sensitive to increase of tension as a dense fluid,

so that an excessive growth at any part, however remote, increases the tension in many directions, and in directions which tend to resolve themselves towards the sinuses and towards the base where lie the cavernous sinuses. In considering this effect of tumours we have, however, to take more carefully into consideration the rate of the growth of them. It may well be that 'optic neuritis' disappears in the course of a case when the tumour is not decreasing in size (β). I think this is not common, but I have seen something like it in more than one instance. It is quite conceivable that the parts may find some new accommodation which is too subtle for plain description; but it is also certain that the growth of a tumour is attended with destructive effects which might soon compensate the encroachment of the tumour itself. Large strands, cut off from their centres, fall into atrophy, as the records of numerous autopsies inform us; and much encephalic tissue falls away from the immediate environs of the tumour, which might soon relieve the increase of encephalic tension. In all this the rate of growth of the tumour must go for much. A rapidly-growing tumour would compress the surrounding tissues and greatly increase tension, which would become specially manifest in the disks on account of the multiplying action of the sclerotic ring (δ); while a slowly-growing tumour would rather suppress the surrounding tissues, cut off their nutrition, and separate them from their connections; making thus a bed for itself at the cost of tissues not displaced but abolished. Rapidly-growing tumours, or tissues of more rapid growth in sluggish tumours, would thus appear to be attended with 'storms' which cannot be properly expressed as symptoms of irritation; not, that is, in the commonly accepted sense of the word 'Reiz.' Headache, for instance, which is mentioned by Benedikt as a chief instance of 'reizung,' may be caused quite as well or better by stretching of the membranes or tentorium (γ), and by pressure upon other sensitive parts. This view seems to me better to explain the great frequency of headaches in cases of intracranial tumour than the vasomotor hypothesis, according to which headache should occur with equal frequency in 'atheroma of

the cerebral arteries,' in acute softening (λ), and many other diseases.

That the mechanical hypothesis suffices for certain cases only (ϵ) is very true, but if proven for some cases there is a presumption in favour of its probability in others where the phenomena are identical. The vasomotor hypothesis, on the other hand, is proven for no particular case, however probable it may be for all. The advocates of the mechanical hypothesis do not, however, wish to have it said that this hypothesis suffices for all cases. They say that intracranial affections are accompanied sometimes by a peculiar swollen and cedematous state of the disks (*Stauungs-papillæ*), sometimes by a neuro-retinitis which presents interstitial inflammation of the disk and neighbouring retina with less swelling; they contend that these two conditions may in many cases be distinguished at once, that in many others a little care and practice will lead to the distinction, and that in all cases the distinction exists, whether evident or not. They contend further, that no one can adequately discuss the question who does not bear this fundamental distinction always in mind, for they say that the effects being different the causes must likewise be different, and that no single hypothesis, whether vasomotor or other, will serve for both results. Benedikt, though not unaware of the distinction, does not keep it before him, and he speaks as if descending neuritis was a defence to fall back upon when the mechanical theory was stormed, rather than a separate fortress, having a garrison of its own. Whether the neuritis does descend or does not (ζ), one thing is certain enough, namely, that in the one class of cases the disk alone is affected, and in the other class of cases the length of the optic nerve is in a state of active hyperæmia with cell proliferation. This has been shown repeatedly in autopsies not only by myself, but also by Mr. Hulke in England, and oftener still in Germany by such men as Virchow, Schweigger, Sämisch, Horner, Liebreich, and many others. Relying upon the vasomotor theory, however, as adequate to explain both sets of events, Benedikt is the more ready to demolish not only the *Stauungs-papilla* but neuritis de-

scendens also. It is difficult to understand the inconsistency which makes him roundly assert on page 250, that 'frequently extreme neuro-retinitis progresses for a long time without any disturbance of vision whatever ("ohne irgend welche Schstörung"), or with a disturbance so slight that the patient does not draw attention to it;' and argue, on the other hand, that there can be no neuro-retinitis, even in a short length of the nerve within the skull, without disturbance of the visual function. Surely if vision coexists with the inflammation of optic nerves in their whole lengths, it may well escape when lesser lengths are involved. Moreover, this argument cuts both ways, for the nerve conduction, if affected by a known pathological change at all, must still be affected by that change, however induced. As a matter of fact, clinical experience and microscopical investigation have led me to believe that severe neuritis may, and often does, exist in the optic nerve without destroying the continuity or the conducting power of the fibres. The mischief rages around them, and although the pressure of new products may compress the fibres and disturb them if these products be in great quantity, yet, generally speaking, their quantity is not so great, and the fibres keep their way unbroken. Thus the mischief descends, and becomes neuritis of the disks before sight is affected. Here, however, a new condition comes into play. The neuritis now becomes neuro-retinitis, a belt of retina of more or less size is involved and its transparency lessened, while over a larger portion still we may note an excess of venous blood and a tendency to cedema, which must seriously interfere not only with the transmission of light, but also with the function of that most delicate structure. We do now often find some loss of vision, be it more or less, we see how it is that its descent from the encephalon may be unperceived, and also why it tends to break up vision as soon as it interferes with the end organ. That neuritis is seen in many cases in which the central irritation is not in the course of the optic fibres (η) is no doubt true, but it cannot be too carefully remembered that neuritis is not so much a disease of nerve as of nerve investment, and that it would be hard to say when the central

irritation is remote from that framework of delicate connective tissue which carries the optic nerves, and other encephalic nerve masses, in one scaffolding. The cause of the sudden onset of blindness in some cases of neuritis (θ) is as yet unknown, or known but obscurely; it seems to me, however, that inflammation may attack the nerve and retina as suddenly as it may attack a lung or a peritoneum, which are a thousandfold greater. The fourth objection, that neuro-retinitis so often occurs in connection with a non-inflammatory intracranial disease (ι), carries no more weight than the objections which have preceded it. Here we need the experience of one actually practised in the use of the mirror, and who will keep us closely to the distinction between ischæmia papillæ—the signal of intracranial tension—and neuritis, the signal of intracranial irritation. Is it the fact that neuritis, properly so called, does coexist so frequently with non-inflammatory intracranial disease? I here join issue, and declare that it does not, but that non-inflammatory disease with increase of bulk gives rise rather to the choked disk (ischæmia); while, on the other hand, primary neuro-retinitis rather accompanies disease, not necessarily attended with great increase of bulk, but certainly attended with irritative proliferations, such as syphilitic meningo-cerebritis. That the curious periodicity of the other symptoms of nerve disturbance is due to vascular storms ($\kappa \lambda$) seems to me not only unproven but of little value if true, for if we have still to ask the reason of the periodicity of the vascular storms, we seem to be no nearer the explanation we desire. If we explain the periodicity of nervous symptoms by assuming that nerve growth, or accumulation of nerve force, consists in the building up of highly complex molecules, which molecules in the normal state have a relative stability, but which in the diseased state have a relative instability, we see, though in a glass darkly, how it is that the nutrition of the unstable parts only reaches a certain elevation, at which the parts then collapse with release of energy. The greater the instability, the sooner this degree is reached, the more frequent the resolution of the molecules, and the less the force released; all of which fits in well with our clinical

experience of deferred and violent convulsion as compared with repeated and diminished convulsion, the latter signifying worse things than the former. On the other hand, to explain nerve instability by vascular instability, which vascular instability must be referred back again, I suppose, to nerve instability, leaves us where the native was left who proposed to escape from the bear by running up a tree and then pulling up the tree after him. In spite of our ingenuity, we find the dilemma remains as unavoidable as ever. The remainder of the quotation from Benedikt (μ) contains little more than a dogmatic reassertion of his position. He points out, that as local intracranial disease is attended by disturbances of remote parts of the encephalon which are due (may be due?) to the intermediation of vasomotor fibres, neuro-retinitis is therefore due likewise to vasomotor irritation. All this is the proof of *incertum per incertius*. That dementia commonly accompanies tumours of the pons, and is therefore due to vasomotor irritation taking effect in the grey matter of the hemispheres, may be the case; at any rate, I am not concerned to deny it. But I must protest against his assumption that hydrocephalus has any such causation. The explanation I have given (p. 159) of the concurrence of secondary hydrocephalus with intracranial tumour is assuredly the true one, and the ventricular dropsy which follows tumour is no more due to vasomotor changes than is the ascites which follows cirrhosis of the liver. As a matter of private opinion, I believe that the secondary affections which follow intracranial tumour are due to many causes, chiefly to distension of fibrous structures, to narrowing or degeneration of arteries, and to direct irritation of nerve nuclei, cells, or fibres. Finally, I would enquire what Benedikt means by 'active hyperæmia' ('eine starke active hyperæmie')? (μ). By an active hyperæmia I should mean an arterial dilatation flushing the fundus with bright blood, as we see in some cases of retinal congestion from exposure to bright light and the like. Such a hyperæmia may well be attributed to vascular palsy, and to vascular palsy it is probably due in the cases to which I allude, but I have never seen such a state as this in any

mode of ischæmia papillæ or of neuro-retinitis, and I am tempted to presume so far as to deny its occurrence.

Such are my objections to Benedikt's explanation of the symptomatic changes in the eye; let me now sum up the arguments for my own side. First of all, let it not be forgotten that, putting atrophy on one side, we have to deal with two kinds of secondary change in the disks: the one is a choking of the disk itself, the nerve and the retina remaining unaffected or nearly so; the other is a hyperæmia with active cell proliferation in the length of the nerve. These two kinds of change have been distinguished over and over again in autopsies, and they are generally to be distinguished during life. In cases which are watched throughout, they are perhaps always to be distinguished; but in the later stages of the choked disk, when the long congestion has impaired nutrition, set up intimate lesions, and favoured cell proliferation, when, in a word, the choked disk has become papillary neuritis, then the distinction which really exists may be invisible to the mirror, and after death more or less of the nerve length may turn out to be involved in the mischief. Still the distinction always remains to the reason, even in cases like that published by Mr. Hulke in the second part of the sixth volume of the '*Ophthalmic Hospital Reports*,' where on the autopsy the choked disk was found, and a descending neuritis also found, which was on its way to the eye. I have described a like case (p. 90), which I examined after death. Now, when we enquire into the association of optic changes with cases of intracranial disease, we find that a large class of them, such, for instance, as encephalic softenings, are not accompanied as a rule by any optic changes at all, although in such cases we have symptoms of wide disturbance, and vasomotor centres or fibres are as likely to be involved as in any other. Nor, again, do we find that tumours in the optic thalami (away from the optic tracts) in the crura and in the pons, which, according to Schiff, contain many sympathetic fibres, are more commonly accompanied with optic signs than tumours elsewhere, but rather the contrary. But we do find optic changes in connection with two kinds of intracranial disease in particular;

the one is tumour, the other is meningitis. Again, when we analyse the matter one degree farther, we ascertain that although the choked disk and the inflamed nerve may coexist with either of these kinds of disease, that, nevertheless, the choked disk is far more commonly found in association with tumour and hydrocephalus than the inflamed nerve. The inflamed nerve, on the other hand, is very commonly found in association with meningitis, and with meningitis, not of the surface nor of parts near any supposed vasomotor centres, but with meningitis near the chiasma. Moreover, it is found to have a special affection for that particular kind of meningitis which forms a part of the syphilitic series, which loves to crawl about the fossæ at the base of the cranium, and which is marked by a peculiarly active proliferation of unstable cells. If now we compare the process of choking the disk with the process of inflaming the nerve, we see that hyperæmia, not 'active' but passive, helped by that multiplying action of the sclerotic ring, which Benedikt also calls to his aid, is adequate to produce the former, while the phenomena of the latter point rather to a biological process of cell proliferation due to propagated irritation. The adequacy of pressure to produce the choked disk is well illustrated by the prominence of the eyeball, not infrequently caused by intracranial tumours, by the ophthalmic symptoms of orbital tumours which often cause swelling and opacity of the disk, and again by the recent experiments of Professor Manz²⁰. By injecting fluid into the cranial cavity of rabbits, he was able at once by the increased pressure to set up dilatation and curvature of the retinal veins with stasis. When we see, then, that pressure is a competent cause of choking of the disk, and when we see, moreover, that the choked disk is commonly found with that particular kind of intracranial or intra-

²⁰ 'Centralbl. d. Med. Wissensch.' Feb. 10, 1870. Compare also a remarkable case of plugging of the ophthalmic vein with consequent venous distension of the orbit, published by Mr. Hulke, 'R. O. H. Repts.' No. VII. 1859, and a like case published by the late Mr. Nunneley of Leeds ('Path. Trans.' vol. xi.): where the same results were found to be due to an aneurism of the internal carotid pressing upon the cavernous sinus.

orbital mischief which increases pressure, are we not justified in requiring very strong evidence before we admit that its causation is quite otherwise? Again, when we know from many preparations in our museums and from clinical experience, that inflammation does tend to run along the course of nerves, as in certain familiar cases of wounds, of contractions of the limbs, intense neuralgias, &c., and when we find such a process as this in the optic nerves, and see that it is likewise attended with the pain due to stretching of its dense sheath, when we see this proliferation also in the third, sixth, or other nerves, not uniformly but according to the accidental disposition of the central mischief, when, again, we find this inflammation of the connective tissue of facial nerves commonly associated with the very similar or identical inflammation of the fibrous structures in the immediate neighbourhood of the nerves, either within the orbit or within the cranium, is not the inference almost irresistible that the neuritis is an offshoot of the meningitis, especially when we discover further, that when this optic or other neuritis, rather than ischæmia papillæ, is associated with tumour, an intermediate term exists in the form of a basilar meningitis set up around the tumour? Such cases are numerous, and very difficult to set aside. When, on the other hand, we look at the arguments against us, when we find them resting in great measure upon conjecture, or upon such observations as those of Adamük, which, however valuable in their bearing upon iridectomy, glaucoma, and the like, seem to bear little upon neuritis and ischæmia; when we find, moreover, that whatever is really known of implication of the sympathetic is against its action in the way supposed; when, for instance, in cases of diseased cervical sympathetic, or of diseased trigeminal ganglion, we find its implication threatens or destroys the coats of the eye without any special implication of the optic disks; when, again, we see in symptomatic affections of the disks that two distinct morbid events need a duality of causation; when, finally, we see in disks so affected that no rosy blush suffuses the nerve and retina, as in the cases of so-called active hyperæmia, but rather a slow, accumulating venous stasis tending

to proliferation ; when we see these weaknesses of the vasomotor hypothesis, we must surely hesitate before we relinquish those older hypotheses which seem to be satisfactory.

For the purposes of this volume, then, I shall still assume that there is such a thing as descending neuritis, and that it is due to propagation of irritation, using that word in the sense in which it is used by the Berlin school. Secondly, that there is also such a thing as choked disk, *Stauungspapilla*, or *ischæmia papillæ*, to be distinguished from primary neuritis, and to be attributed to increased intracranial tension. I believe these two hypotheses explain more facts than any others, and therefore are the best to work with at present. At the same time, I cannot conceal from myself their difficulties,—difficulties which I am not altogether able to remove. These difficulties apply more particularly to the occurrence of neuro-retinitis in connection with tumours, though some questions might arise in respect of the causation of the choked disk also. To take the latter first, it may be objected that, as in the case adduced by Benedikt, and quoted by me in the Appendix (No. 93), a very small tumour remotely situated could scarcely so increase intracranial tension as to interfere with the ebb of venous blood. I would take leave to point out here, however, the very fragmentary and partial state of our knowledge. Benedikt speaks of the optic symptom as ‘neuro-retinitis;’ but Dr. Rydel’s description, although he too speaks of ‘neuro-retinitis,’ is nevertheless suggestive to me rather of the choked disk, and when I look at the *post-mortem* signs, I do not find them to be unlikely companions of the choked disk. But a preliminary difficulty meets me in this, that I am rather disposed to think the growth of the tumour was subsequent to the disappearance of the optic changes, which themselves may have been due to some other cause. If not, its site is such as to lead me to think it probable that it might have pressed upon the great veins of Galen, or upon the lateral or straight sinuses, and thus induced dropsy of the third ventricle, with pressure on the chiasma or cavernous sinuses ; or it may have closed up the foramen of the fourth ventricle, and thus caused ven-

tricular dropsy by blocking the outlet. The state of the third ventricle is not described, but the lateral ventricles contained half an ounce of serum, and their lining was thickened in a way which is suggestive of past effusions.

Unless, then, the advocates of the vasomotor hypothesis are prepared with cases which bear examination better than this one, their arguments must fail. Our examination of this case is one more instance of the impossibility of reasoning on this subject, unless minute care be taken to distinguish between the choked disk and neuro-retinitis,—a distinction which may be difficult or impossible during life, but which is always possible after death²¹.

It is less easy to explain the connection of neuro-retinitis with tumours. To those who are satisfied with such an explanation as this, that the inflammation of the nerve tissue is due to the irritation of a 'foreign body,' I need say no more; but this does not satisfy me, and I fear some of my readers will likewise see a gap in this comfortable reasoning. As a matter of fact, I must admit that I have not found cerebritis around the few embedded tumours which I have been able to examine from this point of view. I believe that tumours as a rule do not set up cerebritis to any marked degree. A little fibrous thickening or capsulation immediately around them there may be, but not any actively proliferating process extending widely through the brain matter²². I have found nothing of this in my own experience, but only the usual products of demolition. At the same time, it is equally true that in these particular cases I did not meet with neuro-retinitis, so that if I have not discovered the mode in which neuro-retinitis might have been caused, at any rate I have not plunged any more deeply into diffi-

²¹ After opening the head, the intraorbital portion of the optic nerve and the disk may be easily obtained by raising up the orbital plate. No disfigurement of the eye need follow this operation, if carefully done.

²² Dr. Maudsley says, in his '*Physiology and Pathology of Mind*,' p. 388, 'If there is one thing which pathological observation plainly teaches, it is the slight irritability of the adult brain. The gradual growth of the tumour allows the brain to accommodate itself to the new conditions, and a closely adjacent nervous centre may be entirely undisturbed in function until the morbid action actually encroaches upon it.'

culties. On the contrary, these negative instances seem to support the opinion I hold on clinical grounds, that genuine neuro-retinitis is rare in cases of uncomplicated tumour, and that the optic change which tumours induce is rather ischæmia papillæ. One case only of neuro-retinitis—where there was a wide lilac-grey patch of inflammation embedding the vessels and involving the deep layers of the retina, but without steep protrusion—I have seen in connection with intracranial tumour, and in this case the tumours (Appendix, No. 81) were situated in the anterior fossa, and the membranes of the base were inflamed and thickened in the pituitary region in such a way as readily to explain the descent of such a process to the eye, which we also verified. Gräfe, who has accurately and minutely studied this matter from the present point of view, refers true neuro-retinitis unhesitatingly to meningo-cerebritis. His words are so important, and I had almost said so conclusive, coming as they do from so cautious and so successful an observer, that it seems desirable to give his words in full, as they appear in his well-known memoir on the subject published in 1866 in the '*Archiv. für Ophthalmologie*,' vol. xii. part 2, p. 114. After saying that neuro-retinitis rarely appears idiosyncratically (comp. p. 74), but rather as a consequence of orbital or intracranial affections (organic or circulatory), he refers briefly to his former distinction, made in the seventh volume of the same journal, between the neuritis which is confined to the disk, and 'marked especially by venous stasis in the papilla, that is to say, by an intense redness, a steep prominence, and often also by hæmorrhages,' and the other form of neuritis, 'in which the swelling and the redness of the disk are less intense, whilst the opacity of the tissue is more pronounced, more extensive, and propagated more deeply from the internal layers of the retina to the middle and outer coats.' This latter form he refers to descending neuritis, and this supposition of propagated irritation passing along nervous tracts is so important, and has so direct a bearing upon the progress of intracranial mischief of many kinds, that I have deemed it necessary to dilate more at length upon the arguments for and against it than may at first seem desirable to the reader.

The following passage, however, I must translate from Gräfe word for word:—‘The supposition which I then made known concerning the existence of these two different types, is in fact confirmed. In the first place, I have had the opportunity on several occasions to make autopsies in cases of the choked disks (“Stauungs-papillæ”). I found in almost all the cases intracranial or orbital tumours. The neuritis was strictly limited by the cribriform plate, and the changes corresponded so exactly to the results published at the time²³, that I need not give any detailed account of them. On the other hand, I have only had three opportunities of examining the other form, and each time I recognised at the autopsy the descending neuritis diagnosed during life. I have already spoken briefly of the first of these cases at a meeting of the Ophthalmic Society of Heidelberg²⁴, but as the two other cases intimately resemble it, I will return to it in a few words.

‘During life there were vague and ill-defined symptoms, pointing to an organic affection of the encephalon, but not allowing of any exact diagnosis. We were, however, in a position to say that a violent encephalo-meningitic irritation was present (symptomatic of a tumour?). Some months before death, a progressive amaurosis declared itself. The mirror showed the slight prominence of the disk, its discolouration, its greyish aspect lightly tinted with red, and, moreover, a diffused opacity of its tissue which extended five millimetres upon the adjacent retina, and then gradually died out beyond. There were also some small hæmorrhages near the disk, the arteries were diminished, the venous trunks, partly hidden by the opacity of the tissue, were thickened and tortuous; but there was no trace of that extraordinary development of their minuter branches, of that intense redness and that prominent swelling which characterises the choked disk. I diagnosed a descending neuritis, and I inferred the presence of inflammatory centres in the brain

²³ That is in the former essay, quoted in ‘Archiv. f. Ophthal.’ vol. vii. Cf. et Koster, ‘Jahresb. d. niederländischen Augenhospitals’ (1865), pp. 8–18, on a remarkable case of *echinococcus cerebri*.

²⁴ Reported in Zehender’s ‘Klinischen Monatsblätter’ (1864), p. 73.

and membranes without marking out their position, but I decided against the supposition of an excessive increase of intracranial pressure. The autopsy showed that the cause of the malady was the presence of certain entozoa (Virchow reported that they were not echinococci, as we first thought, but rather resembling *cænuri*²⁵), which had set up a basilar meningitis extending to the optic nerves. The anatomical symptoms suggestive of decided and prolonged augmentation of intracranial pressure were absent. The following is the microscopical report of Professor Virchow :—

‘The prominent portion shows a considerable thickening of the limitary membrane, and also a swelling of the end of the optic nerve, in which are large vessels with thickened walls, between which is a very dense fibrous tissue. The calibre of the vessels bears no relation to their size, and in many places their canal is rather contracted; the thickening is due chiefly to the adventitia, which is changed into a compact and almost homogeneous mass. The fibrous elements alluded to, at first sight give the impression of fibres of connective tissue, but on tearing the preparation it is evident that they are altered optic nerve fibres. Most of them are thicker than natural, many are varicose, and present fusiform prolongations of moderate size. This alteration is found also in a portion of the expansion of the optic nerve, but the degeneration of the vessels is limited almost exactly to the disk. There is no proliferation of cells or of nuclei in the interstitial tissue; but this is visible behind the cribriform plate, where indeed the neurilemma of the optic nerves is full of nuclei and of new cells. There is also a peculiar change extending to the middle layers of the retina near the disk. We find there the granules of the two granular layers excessively large and pressed together; the intergranular layer presents thick striations, which are perpendicular to the surface of the retina; and farther, on tearing the microscopical preparation with needles, a number of fine but tough fibres, provided with fusiform and varicose prolongations, are teased out from the

²⁵ I may draw attention to this statement, as Reynolds and Bastian (‘Reyn. System,’ vol. ii. p. 497) doubt the occurrence of *cænurus* in the encephalon.

entire thickness of this layer. In many places there is pigment in the external granular layer.

‘The optic nerve in the whole of its length presents a very considerable thickening of the neurilemma, which is separated from the surface of the optic nerve by a cystoid mass, and which only envelopes it as a detached covering. Besides this “perineuritis,” there are the well-marked changes of “interstitial neuritis” throughout the whole length of the optic nerves. In a word, the result of this examination may be summed up in the two latter morbid states (perineuritis and interstitial neuritis in the length of the nerve), and hypertrophy with sclerosis of the vessels of the disk.

‘In the second case, in which I had also diagnosed a descending neuritis, the autopsy was not made until long after the outset of the malady, when a marked atrophy of the disks had set in. We found a very extensive meningitis at the base of the skull, set up by a circumscribed tumour, a meningitis which had directly attacked the optic nerve trunks. The nerves were diminished in size, their neurilemma thickened, and the nervous tubules were in great part indistinguishable. There were unquestionable traces of interstitial neuritis.

‘The third case—a patient who died in the wards of Professor Griesinger—presented (according to the report of Dr. Heine) a circumscribed ramollissement of the left corpus striatum, in addition to a very extensive meningitis of the base of the skull, and a very well-marked descending perineuritis, accompanied by an interstitial neuritis of the optic nerves. These observations have in every case proved that there is a neuritis which propagates itself along the trunks of the optic nerves up to the disks; the result of the autopsies and the march of the symptoms allow of no doubt that this neuritis follows a descending course, and that it is an offshoot of the encephalitis and of the meningitis.’

Such are the thorough and complete observations of Gräfe and Virchow, and the reader may be referred also for corroborative evidence to the communications of Horner in the ‘*Klinischen Monatsblättern für Augenheilkunde*’ (1863), pp. 71–78,

of Fischer in the same for 1866, pp. 164-169, and of Hutchinson in the 'Ophthalmic Hospital Reports,' vol. v. part 1, p. 107. These are the facts and arguments upon which I found my opinion that neuro-retinitis, or neuritis descendens, is due to meningitis either primary or secondary, or in rare cases to encephalitis, and that ischæmia papillæ is due to increase of intracranial tension or to some special interference, such as direct pressure or thrombosis, with the circulation in the cavernous or the petrosal sinuses.

Atrophy of the optic nerve, not consequent upon neuritis, but primary, is a third form of mischief found in connection with intracranial tumour. My own opinion is that primary atrophy results for the most part from the crushing or dividing action of tumours of the base, which sever the nerves from their central connections, from pressure of hydrocephalus upon the optic nerves, tracts or centres, from direct implication in the softening which remoter tumours set up around themselves, or sometimes from propagation of a sclerosis. It is possible, however, that atrophy may also be set up by tumours pressing upon distant but related strands or centres, and so causing optic atrophy indirectly. Lancereaux²⁶ holds this view very strongly, and makes out a good case by careful study of instances in which secondary atrophy of several great strands, including the optic nerves, followed central lesions. The labours of Schroeder van der Kolk and Türk²⁷ on this subject are well known, and of great interest. Charcot and Vulpian have also published very interesting observations of the same kind, and it may be taken as proved that secondary atrophy of the motor strands of the pons, medulla and cord do follow serious lesions, such as softenings and tumours, of the motor centres. The principle upon which these changes are to be explained, was laid down by Waller

²⁶ See his essay upon Amaurosis, 'Arch. Gen. de la Médecine,' Jan. and Feb. 1864.

²⁷ Schroeder van der Kolk's work originally appeared in Dutch, and found its way into several English journals. Türk's principal essay is entitled 'Ueber secund. Erkrank. einzelner Rückenmarkstränge,' Wien, 1851. Gubler has dealt with the subject in a good article in the 'Archives de Médecine' (1859), vol. ii.

many years ago, who first demonstrated the atrophy which results from the severance of nerve fibres from their central connections²⁸. M. Lancereaux finds the explanation of the concurrence of optic atrophy with lesions of the hemispheres, in Gratiolet's supposition, that the hemispheres are their actual centre, a supposition which is far from proved. I must speak with the highest respect for the ingenious and laborious essay of M. Lancereaux, but, as a matter of clinical experience, I find that primary atrophy is generally due to mischief at the base, or to ventricular dropsy, which compress and sever the nerves or tracts at some point in their direct course, that is, no higher than the quadrigeminal bodies. Moreover, the rarity of amaurosis in cerebral softening compared with its frequency in tumour, and, again, the appearance of ischæmia rather than atrophy in tumour of the higher ganglia, seems to me opposed to a belief that the amaurosis is in these cases a Wallerian atrophy.

Nevertheless, there is evidence to make it very probable that severance at the base is an efficient if not the common cause of atrophy. No one has shown more carefully how distinctly such secondary degeneration resembles the atrophy of amaurosis than Dr. H. O. Barth, who in a recent paper has added much to the labours of Türk and Vulpian²⁹. Dr. Barth establishes the truth of the observations of his predecessors, and points out that the degeneration is not primarily of a fatty kind, except about the walls of the larger vessels, nor, again, is it an active nuclear proliferation, but something between the two. He finds the nerve sheaths beset with numerous albuminoid granules, and a great increase of very fine fibres, which make a close network. These seem to be connected with independent nuclei and with the nuclei of the capillaries. This is clearly, he says, an increase of the neuroglia.

²⁸ I ought, however, to admit here that Waller believes the retina to be the trophic centre of the optic nerve, nor is he alone. May not its nutrition depend upon the reaction between its origin and its end organ?

²⁹ 'Ueber secundäre Degeneration des Rückenmarks. Arch. d. Heilkunde,' s. 433, taf. viii. I may explain here that Vulpian and others have shown that the degeneration of a severed nerve takes place pretty equably throughout the peripheric portion. This agrees with my experience of optic nerve atrophy.

Let us now see how far these ophthalmoscopic signs, and the inferences they justify, will help us when we approach the diagnosis of intracranial tumours in detail³⁰. Many symptoms must be taken into account if we are to satisfy ourselves on these points, and although we have to do with the ophthalmoscopic symptoms in particular, yet it will help us in some measure to keep our eye upon some other symptoms also. I shall now divide the intracranial cavity into certain arbitrary regions, in order that we may know what symptoms follow the growth of a tumour in any one of these. Those divisions which I have found most convenient are as follows:—

- (1) Tumours of the convex surface.
- (2) „ whole hemisphere.
- (3) „ anterior lobe.
- (4) „ middle lobe.
- (5) „ posterior lobe.
- (6) „ corpus callosum.
- (7) „ corpus striatum and optic thalamus.
- (8) „ crus cerebri.
- (9) „ cerebellum.
- (10) „ crura cerebelli.
- (11) „ corpora quadrigemina.
- (12) „ pons varolii.
- (13) „ medulla oblongata.
- (14) „ anterior or ethmoïdo-frontal fossa.
- (15) „ middle fossa.
- (16) „ posterior or cerebellar fossa.
- (17) Miscellaneous intracranial tumours.

The nature of the tumour, cyst, or thickening, cannot, as I have said, be distinguished by clinical methods; if in some cases it may be guessed at, the optic signs would give little assistance. It would be impossible even to assume, from the presence of neuritis rather than ischæmia, that the tumour was of a proliferating or irritative kind, as inflammatory action may be the result of lesion from pressure, or pressure

³⁰ Intraorbital tumours are left out of the question for the sake of brevity.

again may be the result of inflammatory accumulations. One remark, however, I may make on the effects of a certain kind of tumour, which is, that intracranial aneurisms seem to interfere less with the special senses than do other tumours. This conviction had forced itself upon my mind in consequence of an autopsy upon a case in which an aneurism of the internal carotid, the size of a cherry and close to the chiasma, had not been attended with ophthalmoscopic signs. I have also seen one or two such cases recorded by others, and I think it must have struck most of us that intracranial aneurisms seem to grow somewhat innocently until their final and terrible catastrophe. I am glad, therefore, to find that Ladame distinctly expresses the same opinion; he says (p. 29), 'aneurisms seldom give rise to symptoms of irritation.' And again, 'Aneurisms, whose seat is especially at the base of the skull, have symptoms which are very analogous with those of tumour in this region. It is remarkable, however, that they rarely give rise to disturbances of the senses.' The explanation of this peculiarity lies, I believe, in this, that an intracranial aneurism is far more elastic and yielding than a tumour. Aneurisms in the head are often little more than thin walled bags, which probably vary much in their states of distension and collapse. The chief exception to this is the tendency of aneurisms of one anterior cerebral artery to cause amaurosis of the one corresponding eye, a tendency which is a very important element in the diagnosis of such cases.

(1) *Tumours of the convexity.*

It is very difficult to isolate the effects of tumours in this situation upon the optic nerves. Tumours of the convexity tend rapidly to destroy the substance of the brain and to affect the mesocephalon, including the thalami, the corpora geniculata, and the corpora quadrigemina. Moreover, fungoid and other tumours springing from the dura mater are very likely to interfere extensively with the cranial sinuses, and thus indirectly with the ophthalmic vein. Granting, therefore, that neuritis accompanied a tumour of the convexity, it would remain doubtful whether the optic change was set up

by direct interference with the ebb of blood, by destruction advancing into the mesocephalon, or by the direct effects of the tumour upon the surface of the hemisphere³¹. The question is one of some interest in this way, that the theory urged by Gratiolet, that optic fibres spread upwards into the hemispheres, and are thus the direct means of cerebral perception of light, is applied earnestly by Lancereaux to the explanation of symptomatic mischief in the disks. He considers, as I have said, that irritation or destruction of certain cerebral attachments of the optic fibres is sufficient to set up secondary atrophic changes in the nerves and disks. Were this so, tumours of the convexity would have much interest for the physiologist as well as for the physician.

As I have said, however, it is very difficult to find a case of tumour of this seat in which the morbid process does not extend deeply into the mesocephalon; so that it is impossible to say how much of the resulting neuritis is due to the superficial mischief, and how much to the deeper mischief. Amblyopia, such as to command the attention of the physician, does not, as a matter of fact, seem to occur in a large proportion of cases of tumour of the convexity. I cannot, however, find any satisfactory case in which the optic nerves were carefully examined during life or after death. Lebert and Ladame found affection of vision in a few cases. Their cases were uncontrolled by the ophthalmoscope; but a lesion of fibres stretching up into perceptive ganglia ought to be indicated very soon by loss of visual function: indeed, loss of visual function should precede obvious mischief in the disks. Cases of tumour of the convexity, however, give no encouragement to the theory that optic fibres pass up directly, and for mere visual ends, into the hemispheres, if I may rely upon the cases collected by myself of tumours in this position, which amount to five-and-twenty in number, and which represent lesions of every superficial part of the brain³². A case is

³¹ This difficulty is the greater, as in so many records of autopsies these secondary points are not observed, or but slightly touched upon.

³² I need scarcely remind the reader that it has been shown by numerous observers, by Voit, Rosenthal, Goltz, and many others, that removal of the hemispheres in pigeons and other animals is not followed by loss of sight.

recorded by Thomas Salter in 'Guy's Reports,' vol. vi. 1841, in which a tuberculous tumour on the surface of the posterior portion of the right hemisphere was attended with amblyopia. Another case, also, I have in my own note-book which was quoted in several of the journals in 1866. It was first reported by Fraumüller. A man, aged 28, suffered from symptoms of tumour, including headache, vomiting, convulsions, &c., and also from loss both of sight and smell. After death a superficial flat (myxomatous) tumour was found lying upon the surface of the right hemisphere. Abercrombie quotes two cases of this kind, both of which were attended with amaurosis; but it is to be remarked that in both these cases the tumour was under the temporal bone, that is to say, near the base of the skull. In one of them, which Abercrombie quotes from Wepfer, there was an exostosis of 3" broad and 1" thick on the inner surface of the left temporal bone; the symptoms in addition to the amaurosis being great headache and convulsion. In the other case, a gelatinous tumour of the size of a hen's egg lay also under the left temporal bone between the pia mater and the arachnoid. There was blindness, together with convulsion. There was also slight right hemiplegia, showing that the tumour affected more than the surface, either by consecutive softening or hæmorrhage, or both. Rosenthal, in his recent volume on nervous diseases, relates a case in which he found several ('mehrere') tuberculous swellings upon the convexity of the left hemisphere. He mentions among the symptoms convulsion and aphasia, with some mental deficiencies, but does not mention any loss of vision. Headache is of course a very common symptom in tumours, which thus directly involve the membranes, and convulsion occurs with the frequency which we notice when morbid processes involve the cortical substance. Mental disturbance is by no means invariable, but undoubtedly common. I find such disturbances in seven out of seventeen of Ladame's cases, but I think this proportion is too small, for I find them in two-thirds of the cases, nine in number, which I have collected in addition. The relation of amaurosis to other symptoms in these rather easily diagnosed

tumours is, then, headache frequent, convulsion frequent, mental derangement frequent, constipation frequent; vomiting seems to be less common than constipation, and amaurosis is occasional, or in simple cases rare. Like fever and strabismus, it is probably a secondary event, and depends directly upon extension of meningitis or softening below.

(2) *Tumours of a whole hemisphere.*

Such tumours might be expected to have only the effects of tumours of any one of the lobes, or of the three separate lobes put together; this indeed may be so, but in the present state of our knowledge it is well to look at the matter from every point of view, and to assume nothing. The size and extent of the tumour has probably effects as important as its locality. Defect of vision is certainly numerous in tumours which occupy all three lobes. Both Lebert and Friedreich found this defect in about two-thirds of their cases, Ladame in one-half; but Ladame thinks his proportion is probably too low. When we see that disturbance of vision forced itself upon the notice of the physician in so large a number of cases, we may well suppose that a far greater number would have revealed ophthalmoscopic change. Take, for instance, the case quoted by Abercrombie from De Blois, where three tumours were found in the right hemisphere in a boy aged seven. In this case, although hydrocephalus was also present, yet no affection of vision is mentioned. The intense headache probably concealed any dulness of vision which may have existed, and my experience will scarcely allow me to doubt that marked changes might have been found by the mirror, not only in this, but in many other cases, of tumour of equal extent. The case occurred in 1821, long before the ophthalmoscope was thought of.

A case is recorded by Powel in the 'Medical and Chirurgical Transactions,' vol. v. p. 219, in which weakened vision, dilated pupils, and finally blindness occurred in a case where a tumour was found with three other hardened portions (gliomata?) in the upper part of the length of the right hemisphere. In some of the similar cases I have collected there

was hydrocephalus, in others there was none. The careless way writers have even yet of including the corpora striata and the optic thalami with the hemispheres, and also of calling all the nervous centres in the head together the cerebrum or brain, makes it difficult to be sure, in particular cases, whether the mischief did or did not penetrate below the ventricles, or set up secondary changes below them. One thing, however, does seem clear, that while in superficial adventitious growths upon the convexity, loss of sight is rarely present, in tumours which occupy the mass of one hemisphere affection of the optic disk must rarely be absent. We should still, of course, expect to find convulsion a common feature in disease so deeply involving the cortical substance; while hemiplegia would be less common, and would occur only when the mesocephalon was compressed, softened or involved. Such is the conclusion derived from the cases themselves, the relation of symptoms being—headache very common, convulsion very common, mental deterioration, amaurosis, and constipation common; vomiting and hemiplegia not so common. A young lady whom I saw once or twice with Mr. Teale presented this array of symptoms with fine choked disks. After death a tumour was found occupying almost the whole of the right hemisphere. We diagnosed tumour of the hemisphere but had nothing to tell us of its extent. Fever is rarely mentioned, and probably occurs only in company with meningitis. Other nerves of the base unaffected unless by extension of meningitis, which seems to have been the cause of ptosis or strabismus in one or two instances.

I may perhaps remind the reader that recent observers are disposed to attribute emotions, if there are such things, rather to the right hemisphere. Certainly I find emotional disturbance to have been more common in disease of the right brain.

(3) *Tumours of the anterior lobes.*

Dr. Russell Reynolds, in his 'System,' page 483 (ed. 1868), thinks that 'convulsions are most frequent in tumours of the cerebellum, and that they diminish in frequency as the seat

of lesion advances forwards, i.e. through the posterior and middle to the anterior lobes of the cerebrum; and that amaurosis, impaired articulation (speech?), and intelligence observe a contrary relation to those lobes, being most common when the tumour is in the anterior cerebral lobes, and relatively less frequent as the seat of the tumour retrogrades.' I have myself only met with the one case of tumour in an anterior lobe, to which I have already made allusion (App. No. 81); but I have collected records of thirty-eight well-marked cases. In nine of these only are dimness or loss of vision noted; but of course ophthalmoscopic signs may often have been present though no loss of vision was observed. Some of these cases are well reported, and my impression from these well-reported cases is, that optic signs will be found to occur frequently in tumour of the anterior lobes. Consecutive mischief soon reaches the pituitary region, as in my own case, and sometimes the morbid growth penetrates the orbital plates. I have not included these latter cases, however, as disturbance of the eye must occur in them as a matter of course; and they are, from our present point of view, to be called orbital rather than encephalic tumours. Dr. Todd described an interesting case in the 'Medical Times' of 1853 (vol. ii. p. 166), where a large hard tumour in the anterior lobe of the right hemisphere caused convulsion and amaurosis without paralysis. The optic tracts were softened, and the corpora geniculata were of an abnormal colour. The optic nerves examined under the microscope presented numerous oil globules. It seems *a priori* very likely that progressive softening surrounding tumours of this region should invade the optic tracts and nerves, and thus cause primary atrophy; but at present our ophthalmoscopic evidence is scarcely sufficient to bear this out. Lancereaux, in the valuable essay to which I have referred, on 'Amaurosis in connection with degeneration of the optic nerves in cases of alteration of the cerebral hemispheres,' publishes an admirably described case in which a serous cyst of the right anterior lobe was found, with atrophy of the geniculate bodies, optic tracts, left cerebral peduncle, and pyramid, with the corresponding column of the cord.

There was no hydrocephalus. Lancereaux is convinced that in this case there could not have been any pressure upon the sinuses at the base. In a second case, recorded with equal minuteness, a neoplasm the size of a duck's egg was found in the left anterior lobe coexisting with softening of surrounding parts, alteration of the optic tracts and nerves, and slight atrophy of the left cerebral peduncle and pyramid. In this case, however, there was hydrocephalus, and the corpora quadrigemina were compressed³³. The symptoms were headache, dizziness, convulsions, amaurosis; no palsy of movement or sensation. With the mirror Liebreich found the left disk pushed forward and greyish; arteries thin and pale; veins swollen and dark; right disk less prominent and white, and falling into atrophy; vessels almost lost. Ladame, again, gives an interesting case recorded by Nolte, '*Med. Zeit. f. Heilk.*' No. 38, 1835, in which 'oscillating amblyopia' was found to depend upon the pressure of a distended third ventricle upon the chiasma. The ventricular dropsy coexisted with a tumour in an anterior lobe the size of a duck's egg. It is unnecessary for me to add to this section; in the vast majority of cases recorded the mirror was not used; while, on the other hand, those who used the mirror record only positive instances. I have said enough, however, to show that amaurosis does often coincide with tumours of the anterior lobes, and signs visible with the mirror occur no doubt, much oftener, as we see from case No. 82 in the Appendix, where Dr. Jackson found optic neuritis in a patient who was able to read. After death a tumour was found in the left anterior lobe. The amaurosis seems to be due, in some cases to ventricular dropsy, with pressure on the chiasma, the tracts or the corpora quadrigemina, in which cases we should probably see atrophy as the first morbid change in the disks; in other cases, as in the second of Lancereaux, the choked disks were found which were due to pressure upon the basal sinuses, probably direct. In three cases I find records of the tumour with the brain matter below it having

³³ Such was the case also in an example which I extracted from Virchow's '*Krank. Geschwülste*,' p. 662-3; and in which lessened visual power coexisted with a tubercular tumour of the right anterior lobe.

caused atrophy of the optic nerves by direct pressure; and, lastly, it seems likely that atrophy of the optic nerves may result from the extension of softening in their direction, this softening not being due, I think, to implication of optic fibres in the hemispheres, as Lancereaux would have it, but to mere extension by contiguity to the nerves, tracts, or corpora quadrigemina, or more frequently to pressure upon their blood-vessels. I have no evidence that neuro-retinitis occurs in these cases unless meningitis be present. The summary of the other symptoms of tumour of the anterior lobes seems to be—headache (frontal or general), mental derangement, convulsions, aphasia (if the mischief be in the left brain), and anosmia. Hemiplegia and disordered sensation are absent, unless softening or pressure extend to the mesocephalon.

(4) *Tumours of the middle lobes.*

I have seen no case of tumour confined to the middle lobes. Ladame enumerates twenty-seven cases, to which I have added notes of four others. In several of Ladame's cases, however, the mischief involved the rest of the hemisphere also, or involved the mesocephalon. Ladame only mentions hydrocephalus in two cases. External squint, as we should expect, now becomes more common, and occurs in about one-fourth of the cases. Ladame records amaurosis or amblyopia in seven out of twenty-seven cases. Galezowski records very carefully a case in which a fibro-plastic tumour of the right middle lobe caused amblyopia, and after death was seen to compress the mesocephalon forwards and downwards. The thalamus, tract, and geniculate body were also crushed. Indeed, even the bones at the base were injured, so that the case may be considered too extreme a one for our present purpose. The size of the tumour is not mentioned. As hydrocephalus does not seem to be a very frequent companion of tumours in this seat, amaurosis, when it occurs, is probably due rather to the direct action of the tumour in the way of pressure or softening. In Case 84 in the Appendix, however, an ounce of fluid was found in the ventricle of the opposite side. In

most of Ladame's cases in which amaurosis is mentioned, compression of the optic nerve is recorded among the post-mortem discoveries. We have no means of knowing, as yet, how often the mirror would reveal congestive changes in the disk in tumours of the middle lobes, but probably much less often than in tumours originating at the base. I publish one of my own cases in the Appendix (No. 84) in which optic neuritis was present.

The nearness of the mesocephalon explains the frequent occurrence of palsy of motion and sensation in cases of tumour of the middle lobes. Ladame found hemiplegia in half of his cases, and slighter palsy in many more; disturbed sensibility he found in ten cases. I have a curious case now under my care in which many good observers accept my diagnosis of tumour of the right middle lobe with the thalamus, and in which there is some weakness of the left side, but more especially a curious degree of anæsthesia. When his eyes are closed, the endeavours of this patient to pick up anything upon the table are very odd failures; indeed, he often has the object in his hand without being aware of it, and continues his groping efforts. There are no ophthalmoscopic signs. Headache in the parietal region is generally present, but psychological derangement is absent in one half the cases, or even more, at any rate during the earlier stages. On the whole a somewhat greater frequency of amaurosis and the appearance of deranged sensibility distinguishes tumours of the middle from those of the anterior lobes. When the disease is on the left side, there would probably be some frequency of aphasia.

(5) *Tumours of the posterior lobes.*

My own experience seems to be peculiar in the matter of the connection of amaurosis with tumours of the posterior lobes. Of Ladame's fourteen cases amaurosis occurred in two only, and he grudges the symptom in one even of these, as he will have it to have been an accidental complication. I have had, however, one case of tumour of a posterior lobe attended with amaurosis, the heads of which are given in the Appendix (No. 86). I publish there also another (No. 57), quoted

from Dr. Lomax of Philadelphia, which may be considered here though the mischief was probably rather of a sclerotic kind. Gräfe (*loc. cit.*) gives a case in which optic neuritis co-existed with headache and convulsion, and in which a myxoma the size of a small apple was found 'at the posterior extremity of the right hemisphere.' Lancereaux, again (*loc. cit.*), quotes a case (Bainbridge, 'Med. Times,' April 10, 1840) of a child of nine, who had headache, vomiting, convulsions, and amaurosis as consequences of a 'medullary sarcoma' of the left posterior lobe. The corpora quadrigemina were here found softened and pulpy. In a case of hydatid of the left posterior lobe with amaurosis recorded by Dr. Barker in the tenth volume of the 'Pathological Transactions,' no mention is made of the state or position of the mesocephalon. In Dr. Hughlings Jackson's case (Appendix, No. 85) the choked disk coexisted with dilated ventricles. Finally, Galezowski (*loc. cit.*) records a case in which amaurosis slowly came on with other and obscure cerebral symptoms; mischief was found in both posterior lobes, and the corpora quadrigemina were injured, partly indeed destroyed. Amaurosis must, I think, occur more frequently therefore in consequence of tumour of the posterior lobes than is generally said to be the case. When it does occur I am disposed to attribute it in many cases to interference with the corpora quadrigemina, either by means of transmitted pressure or transmitted softening. If this be so, the ophthalmoscopic appearances should be distinctly subsequent to the first complaint of dimness or of blindness, and the only change to be looked for in the disks should be atrophic. In other cases no doubt hydrocephalus is the nexus, as in No. 85. The posterior lobes lie close upon the straight sinus and the veins of Galen, so that ventricular dropsy would soon have reason to appear. Such a case is also published by Bateman in 'The Edinburgh Medical and Surgical Journal' (1805), vol. i. p. 150. The case is re-published in the Appendix (No. 66). That the swelling in this case was an abscess, need not, I think, prevent my quoting it in the present connection. As regards the other symptoms of tumour of the

posterior lobes, we find that psychical disturbance is very common, a clinical fact which bears closely upon some recent speculations by Bastian and others concerning the function of these lobes, and of the remarkable cells contained in their convolutions. There can be no doubt that the mere anterior position of the frontal lobes excites a prejudice in their favour which is unreasonable. Vomiting, again, is commoner with tumours of this seat than when placed more forward. Convulsions also are prominent, as in all mischief irritating the cortical substance. Headache, sometimes occipital, but more often of a general character, is rarely absent. Disturbances of sensation are as rare as in tumour of the middle lobe they are common. Complete hemiplegia is rarely seen in simple cases.

(6) *Tumours of corpus callosum.*

I only have before me the four cases collected by Ladame. The symptoms of tumour of this region seem to be those of tumour of the hemispheres. In no instance was sight affected, nor was it affected in the cases of deficient corpus callosum recorded by Langdon Downe.

(7) *Tumours of corpora striata and thalami.*

It is a curious commentary upon the thalamus opticus, that tumours therein do not affect vision with anything like constancy. If they involve the hinder and outer portion of the thalamus, one would surely look to see the sight affected and the nerves injured, but I think, as a matter of experience, that tumours, like bleedings, rather prefer the forward portion and the striate bodies. Nevertheless, among the large number of cases of which notes are before me, involving the under and hinder part of the thalamus, I certainly find many in which tumours were discovered, and yet no visual defect is mentioned. This curious result reminds us of the fact that hæmorrhage, which so often affects these bodies, has rarely a destructive effect upon vision. Galezowski found defect of vision recorded in seventeen cases out of sixty-two of lesions of the thalami. I have,

indeed, numerous cases before me in which amaurosis was seen in tumour of this region; but many of these are the sort of exception which prove a rule. In one case of amaurosis a tumour was found in the left thalamus, but there was also another in the left lobe of the cerebellum. M. Bouchut, among the cases which, with and without proof, he records as tumours of the encephalon, gives one case with an autopsy. In this case congestion of the disks was noted during life, and three tumours were found in the left striate body and thalamus after death. But there was also a tumour in the medulla oblongata. In this case M. Bouchut found the left disk more œdematous than the right, and from this and other cases he hazards the assertion, that we may in such cases determine the side of the lesion by the side of the chief optic trouble³⁴. I believe there is no good ground for this supposition; in my own experience, accident enters far too much into the genesis of symptomatic changes in the disks to enable us to draw definite conclusions of this kind. But this by the way³⁵. If I analyse further the cases of tumour of the thalamus or striate body which were attended with amaurosis, I come to a case of Friedreich's (*loc. cit.*) in which amaurosis co-existed with a tumour the size of a hen's egg in the right thalamus. Here, however, this careful pathologist notes that the corpora quadrigemina were quite crushed. Another very interesting case is recorded, too, by Leyden in Virchow's 'Archiv.' for 1864 (B. xxiv.); but in this there was pressure upon the vein or veins of Galen, and consequently hydrocephalus was a prominent feature at the autopsy. The tumour was in the left thalamus, and was the size of an apple. In a case recorded by Dr. Johnson in the 'Med. Chir. Rev.' Jan. 1836, there were cysts in the left ventricle beside the mischief in the thalamus; and in a case recorded in the 'Lancet' (1850, p. 682), a carcinoma in the left thalamus not only caused amaurosis, but a degree

³⁴ 'Du Diagnostie des Maladies du Système Nerveux,' p. 318.

³⁵ I may say here, that the many assertions we see in case reports, that one eye alone was affected, have no value unless the mirror was used.

of dilatation of the pupils and of involuntary micturition which suggest that the tumour involved also the neighbourhood of the corpora quadrigemina. I myself recently had an autopsy upon a case of tumour in the right thalamus and corpus striatum, which I had attended in consultation with Mr. F. Hall of Leeds. There were no ophthalmoscopic signs of change during life, but a sarcoma the size of a hen's egg, occupying the right striate body and the anterior half of the thalamus, was disinterred, and it was surrounded by a layer of atrophic softening. The optic nerves and corpora quadrigemina were normal, the sinuses were free. On the other hand, I am now attending a man under the care of Mr. Mann, of Leeds, in whom we have reason to suspect tumour of the right corpus striatum, and he has well-marked choked disks. In the thirteenth volume of the 'Medico-Chirurgical Transactions,' a well-known case of tumour of both thalami with amaurosis is reported by John Hunter, junior; but there was great disturbance of almost all the encephalic functions. Absence of optic changes seems, then, to be common, perhaps the rule, in tumours of the striate bodies and thalami which do not press upon the corpora quadrigemina or the veins of Galen, and this seems to bear somewhat against the vasomotor theory of optic disturbance. Fortunately, there are other symptoms of a more constant kind, in the disturbance of the motor tract. Convulsions are generally present, and contralateral hemiplegia of more or less severity, according to the degree in which the motor fibres are thrust aside or sundered. Some degree of loss of memory and mental application is generally present, and speech is generally affected in some measure, though rather in the way of drawling or defective articulation than of aphasia proper. Vomiting and constipation occur in many but not in most cases, and severe headache is less frequent than in tumours which grow nearer the membranes.

(8) *Tumours of the crura cerebri.*

Tumours of the crus are not very uncommon. I have records of eleven cases before me, and they bear out the

belief that lesions of the crus are easy of diagnosis. The best essay upon the pathology of these cases is by Dr. Hermann Weber, and is contained in the twenty-sixth volume of the 'Medico-Chirurgical Transactions.' His cases, however, were not cases of tumour. In addition to the invariable or almost invariable lesion of the oculomotorius, visual defect is often noted. A case is reported by Mr. Paget in the 'Medical Times' of February, 1855, in which a tumour of an inch in length and half an inch in thickness was found in the right crus of a man aged 41. There was anæsthesia of the right arm, dizziness, palsy of left face, and oculomotorius and convulsions of the limbs of the same side. The sight was weakened³⁶. In a case quoted from Mohr by Ladame, in which amblyopia was found, a tumour in the left crus was seen to compress the corpora quadrigemina, and to have involved them also in a belt of softening.

Dr. Hughlings Jackson, again, records a case (Appendix, No. 88) in which optic neuritis coexisted with a tumour of one crus. In a case lately cited by Dr. Hoffmann, and quoted in several journals, in which a tumour the size of a cherry-stone was found in the left crus, the following symptoms were noticed:—paresis of the left limbs, some anæsthesia of the left face, palsy of the left oculomotor nerve, and amaurosis of the right eye, with atrophy of the nerve. The left eye was affected with glaucoma. On the whole, then, clinical experience bears out what anatomy would suggest, namely, that swellings of the crura which lie so near the optic tracts, corpora geniculata, and corpora quadrigemina, tend often to involve and injure these parts, and to set up a corresponding defect of vision. As regards the ophthalmoscopic appearances in particular, we have only Dr. Jackson's case to guide us; but I am disposed to guess that primary atrophy due to compression or softening of

³⁶ If I may depart from the subject in a note, I would refer also to the incontinence of urine recorded in this interesting case, which reminds us of Budge's assertion, that contractions of the bladder are caused by irritating the crura cerebri.

the visual centres or tracts, would be a commoner result than optic neuritis in cases, that is, of tumour of the crus uncomplicated with meningitis. I have an interesting case under my care at present, presenting complete palsy of the left third nerve with decided and progressing palsy of the right nerve also. For the rest, motion, sensation, and intellect are unaffected. I believe the case to be one of tumour; it is scarcely worth reporting without an autopsy, but I may state that primary atrophy of both optic nerves is present. It may be, however, that the supposed tumour is in the corpora quadrigemina. Tumours of the crus alone are too small to raise intracranial tension, they do not press directly upon the basal sinuses, and they are perhaps more likely to compress or soften the parts of vision than to irritate them, unless meningitis be present³⁷. The other symptoms of tumour of the crus are—palsy of the oculomotorius on the same side (this is almost invariable, and the tumour soon involves the origin of the opposite oculomotor), dizziness (depending more or less upon diplopia), disorders of both sensation and motion³⁸, the latter (if not the former also) being contralateral. Paralysis of the facial on the opposite side is common, and may perhaps be due to disturbance of certain fibres which are now said to pass from the motor ganglia, through the crura, to the nuclei both of the facial and hypoglossus. Weber considers that the vagus is especially involved in disease of the crura; but his opinion is not supported in any definite way by my cases. Headache is less common than dizziness, and vomiting and constipation, though present in some cases, do not seem generally to be well marked. The mental functions are not implicated until quite the later stages of the disease.

³⁷ In one of Dr. Weber's cases (*loc. cit.*) one optic nerve was found to be degenerated in the part which bordered upon the crus.

³⁸ Weber says that sensation is disturbed on the opposite side. This seems, however, as yet uncertain. I may say that I collected a number of cases for publication about a year ago, to prove that lesions of a certain district of the foot of the crus outside the thalamus cause cutaneous anæsthesia. I still intend to publish these cases, but I find that others have said my things before me.

(9) *Tumours of the cerebellum.*

The very common association of amaurosis with diseases of the cerebellum is an old subject of speculation, and some pathologists have been tempted to suppose that the cerebellum is in some way a visual centre. Such is certainly not the case; but, as we shall see, it is by no means easy to explain the reason why disease of the cerebellum so often sets up disease of the optic nerves. That absence of the cerebellum does not include absence of vision, is clear from the well-known cases published by Cruveilhier³⁹ and Combette⁴⁰, in which there was a congenital absence of the cerebellum with preservation of the special senses, and of sight in particular. In these cases the corpora quadrigemina were intact. Two main points offer themselves to us when we seek an explanation of the influence of cerebellar disease upon vision: the first is that no function has as yet been definitely appropriated to the cerebellum; and, secondly, that, isolated as the organ seems to be, yet its troubles have nevertheless a way of making themselves felt beyond itself. I have the records of one hundred cases of tumour of the cerebellum now before me, and I might easily have added to the number, had I thought it worth while. Seventy-seven cases are tabulated by Ladame. The very variety and multiformity of the symptoms in these cases is sufficient to show that no generalization can be drawn from them unless it be of a negative kind, and to show, moreover, that diseases of the cerebellum, when they reveal themselves, do so rather indirectly by setting up secondary disturbances elsewhere.

It seems likely, indeed, that the only symptom which really belongs to disease of the cerebellum itself, is a degree of weakness of the limbs which does not amount to definite palsy. To call the organ a centre of motor co-ordination, is, to my mind, a very thoughtless hypothesis, for surely every point in the body where two nerve-fibres are brought into functional relation, is so far a centre of co-ordination.

³⁹ 'Anat. Pathol.' vol. i. part 15, p. 5.

⁴⁰ 'Journal de Physiologie, par Magendie,' vol. xi. p. 27. 1831.

The cerebellum appears to me to be rather a reservoir of force where, by the means of the posterior columns, tension is stored up during times of repose to be given out during times of demand. Want of capacity of motion rather than palsy, is what we should look for in the loss of such an organ. I must not, however, pursue this point farther now, but I have much evidence in support of my view, derived both from human and comparative physiology. It is sufficient for present purposes to dissociate vision from the possible functions of the centre.

As to the second point, which is the remarkable disturbing power which diseases of the cerebellum have upon the other encephalic centres, I have more to say, as affection of vision is included among these interferences. It is unnecessary for me to relate cases in which tumour of the cerebellum has coexisted with amaurosis, for the coincidence is universally admitted to be common. I publish one case, however, which was kindly sent to me in manuscript by Dr. Roberts of Manchester, in further proof of the fact, sometimes disputed, that amaurosis is often found in connection with diseases of the cerebellum which are not tumours. Dr. Dickenson and others have published similar cases of cerebellar softening with amaurosis.

Among the symptoms of cerebellar disease which are not due to the nerve destruction of its own tissue, but to interference with other parts, are occipital headache, due probably to stretching of the tentorium and membranes; troubles in swallowing, circulation and breathing, due to interference with the medulla; irritation of the genitals, due perhaps to irritation of the same region; convergent squint and dilatation of pupils, due to interference with the nucleus of the sixth nerve and with the quadrigeminal bodies; convulsions, due to interference with the great underlying motor strands below the fourth ventricle; vomiting, of uncertain causation, but occurring rather in cases of congestion and swelling mischief than of mere nerve tissue destruction; and so on to amaurosis, occurring with both kinds of mischief, and the cause of which we have to learn, if we can.

Let us now begin to learn from the other side, and ask in what way the optic nerves betray their disturbance? Unfortunately, in very few cases have we the control of the mirror to satisfy us in this matter. Galezowski (*op. cit.*) gives a case of tumour of the cerebellum with the ophthalmoscopic appearances. The state of the disks seems to have been ischæmic rather than neuritic, though he calls it neuritis, and refers it to propagated irritation by way of the processus ad testes. A tumour was found in the 'antero-inferior' part of the cerebellum upon the petrous bone, and thrusting the left lobe towards the median line. The retina was cedematous and infiltrated, and many little hæmorrhages were found in it. The optic nerves were softened and degenerated, and the chiasma was 'small, greyish, and pulpy.' The state of the ventricles is not mentioned. In a case of myxosarcoma of the cerebellum described by Leber (in the '*Arch. f. Ophth.*' vol. xiv. part ii.), he observed a state of the disks suggestive of the passing away of the choked disks and the commencement of subsequent atrophy. After death the disks were thickened with increase of connective tissue, while the length of the nerves presented only atrophied fibres with fat granules. Atrophy of the disks was complete before death. In the Appendix, again (No. 92), will be found a case of tumour of the cerebellum also attended with the choked disks. Although our knowledge of the appearances of the disks in the early stages of disease secondary to cerebellar tumour is thus defective, yet we have ample evidence that in the later stages white atrophy is very common. At this period sight is more or less lost. Certainly it would seem that primary atrophy—atrophy without blurring or ragged edges—is a very common result, and that in cases of cerebellar tumour, sight is soon affected, and dimness quickly advances to blindness. Amaurosis pure and simple, white atrophy with loss of sight, and probably not preceded by neuritis or ischæmia, seems, if we may judge from clinical histories, to follow tumour in this region more decidedly than tumour elsewhere, save at the anterior base. What we have to account for seems to be, first, primary atrophy; secondly, and less commonly, the choked disk; thirdly, and

probably less commonly still, neuro-retinitis. Now how are we to explain these occurrences? Galezowski, following many other writers, says unhesitatingly ⁴¹, 'We can only attribute the amaurosis in cerebellar affections to the propagation of secondary inflammation up to the optic centres' (*corpora quadrigemina*). At present, we have only to deal with cases of tumour, and in these cases I very much doubt the accuracy of this somewhat positive conjecture.

I have said that in examining the injured tissues which surround encephalic tumours I fail in the large majority of cases to find any evidence of acute proliferating processes, and that I find evidences rather of occlusion of blood-vessels with simple degradation and traces of hæmorrhage. That softening may extend to the *corpora quadrigemina* by way of the *processus ad testes* I think likely, but I wait to be convinced that any inflammation reaches them. Progressive softening, reaching the visual centres, is probably one cause of loss of vision and of nerve atrophy. When we further consider the position of the cerebellum, and remember how closely it is invested by its containing structures, by the rigid occipital box, and by the dense tentorium, we shall see at once that any enlargement of its volume, however slight, must soon exercise strong pressure upon the walls of its box. Pressure upon the occipital bone means pressure upon the lateral sinuses, and pressure upon the tentorium also means pressure upon the straight sinus, upon the outlet of the veins of Galen, and upon the torcular Herophili itself. It is evident, then, that slight enlargements of the cerebellum must very soon interfere seriously with the reflux of nervous blood. Again, if we leave the blood-vessels and turn to the relative position of the soft parts, it is clear that in enlargement of the cerebellum, especially of its median and anterior parts, there must be a resolution of the pressure in the direction of the mesocephalon, as this is the direction of least resistance. Were this found to be the case, we should see the *corpora quadrigemina* flattened, and we should infer that their function was suppressed. In the notes before me, I do

⁴¹ Loc. cit. p. 166.

not find that special attention has been given to this point, though the very frequent mention of 'pupils widely dilated' is suggestive of pressure upon the corpora quadrigemina. As regards the transmission of softening to the corpora quadrigemina, I find in one case (No. 85 of Ladame) that softening of the corpora quadrigemina and atrophy of the optic nerves was found to coexist with a cerebellar tumour of the size of a hen's egg. Amaurosis was among the symptoms. It would seem to me, then, that softening and pressure, rather than inflammation, are the agents of interference with vision. But it is to the interference with the venous circulation that I would attribute the amaurosis in a large number, if not in the majority, of cases of cerebellar tumour. We have seen how this interference takes place, and we shall no longer be surprised to find that ventricular dropsy is consequently found in so great a proportion of these cases. Hydrocephalus is actually mentioned in the majority of cases, and, so carelessly are autopsies made or noted, in many more where no allusion is made to the state of the ventricles, we may suppose that it existed to some degree at least. Now I have already pointed out how large a part is played by ventricular dropsy in causing optic atrophy, and if I turn to my cases of cerebellar tumour I do not lack further evidence on this part. Not only must the great dropsy of the side ventricles have compressed the optic tracts in cases where, however, no examination of the tracts is recorded, but I find in several cases, that the direct pressure of a distended third ventricle upon the chiasma was observed and noted. In case No. 67 of Ladame, for instance, we read, 'loss of vision and third ventricle distended.' In a case published by Bouchut, in the '*Gazette des Hôpitaux*,' No. 144 (1854), we read, 'complete amaurosis,'—'third ventricle distended;' and in a case which came under my own observation, a distended third ventricle had flattened and almost destroyed the chiasma, which was thin, grey, and scarcely consistent. If neuro-retinitis were found in a case which presented the symptoms of cerebellar tumour, I should attribute it to meningitis. I can find no evidence to lead me

to suppose that tumours, although they may be called 'foreign bodies,' set up irritative proliferation which travels along brain substance from the cerebellum to the eye; and, on the other hand, I find that meningitis has been found to coexist with cerebellar tumour in many autopsies, especially when these tumours were superficial. Cerebellar tumours, moreover, are often tubercles, and in these cases a coexistent tubercular meningitis of the base would be likely enough. To sum up, then, the frequent concurrence of amaurosis with cerebellar tumour depends chiefly upon the neighbourhood of the corpora quadrigemina, and of the great encephalic veins and sinuses. Its causation is most commonly due, perhaps, to the ventricular and subarachnoid dropsy, which results from venous stoppage, and which crushes the nervous centres or tracts of vision, or in an early stage chokes the optic disk. In other cases it is due to the advance of softening from the circumference of the tumour along the processus ad testes to the corpora quadrigemina. The optic nerves waste, either as a consequence of the destruction of the visual centres, or because they are themselves compressed. In many cases, where the mischief is due to venous arrest, a period of strangulation may be seen to precede the more complete destruction, if sought for in time. The state of *ischæmia papillæ*, indeed, has no doubt existed in some cases where no disturbance of vision is recorded, and where few or no ophthalmoscopic examinations were made. True neuro-retinitis seems to occur but rarely, nor should we expect it to occur, save when meningitis is present. In cases of tuberculous tumours, meningitis of the base may be often present, and neuro-retinitis may be its consequence. In very superficial tumours of the cerebellum, as of other superficial parts of the encephalon, adhesive meningitis is also a common event, and may, in rare cases, propagate itself to the middle fossa, and so to the optic nerves.

I have already (page 157) enumerated the various other symptoms which must be looked for in connection with the optic signs in cases of tumour of the cerebellum.

(10) *Tumours of the crura cerebelli.*

Ladame gives two cases only of tumour of the pontine crura of the cerebellum, but to these I have been able to add seven more. I make a separate heading of these cases, because in six cases vision was profoundly affected, in two vision is not mentioned, and in one vision was unaffected, but neuritis was discovered by the mirror. In this last case only have I notes of the ophthalmoscopic appearances: the case is published by Mauthner, in his '*Lehrbuch der Ophthalmoscopie*,' p. 293. In the right crus cerebelli ad pontem was found a sarcoma the size of a walnut. There was considerable dropsy of the ventricles. (Vide Appendix, No. 91.) The ophthalmoscopic appearances bear out the conjectures I have previously made concerning the mediation of hydrocephalus in these and like cases, and concerning the effects upon the eye. Although Mauthner uses the expression 'fully-developed neuritis' in describing the state of the disks, yet this was not descending neuritis, for at the autopsy the microscopical examination showed that the mischief was quite confined to the disks, the nerve above and the retina beside being quite normal. It adds to the significance of this striking case, that it is the only case in which vision is reported to be unaffected. Mauthner says distinctly, that the patient enjoyed perfect sight up to the end of his life ('volle Sehschärfe bis an sein Lebensende'). Here, then, clearly we had not to do with pressure on the corpora quadrigemina, or with destruction of these bodies, as was probably the case in some of the other like instances in which amaurosis was present; but we may assume that the ischæmia of the disks was due to the pressure of the hydrocephalus upon the cavernous sinus, or that the ischæmia and hydrocephalus were common direct results of the venous arrest caused by the tumour.

I have notes of three cases of tumour of the superior peduncles alone, and in all three sight was affected; these cases corroborate my remarks in the previous section, to which I need not make any addition. Among the con-

comitant symptoms of lesion of the crura cerebelli come those remarkable lateral movements, or movements of rotation of the head and body, which have been noticed in these, and in some other one-sided lesions, by many observers. Headaches and dizziness also occur, and hemiplegia, which probably depends upon more or less interference with the great motor strands continuous with the crura cerebri.

(11) *Tumours of the corpora quadrigemina.*

Hitherto, in classifying our tumours, we have not been very careful to be sure that they were limited to the part under discussion. In cases of tumour of a lobe of one hemisphere, for instance, we were already pretty sure that it could only affect vision by affecting also some part of the encephalon other than its nominal seat, and we had to learn whether this was commonly the case. Latterly, however, we have had to look more carefully to the exact seat of the tumour, and in discussing the effect of tumours of the corpora quadrigemina upon vision, we have to look minutely to the very place of the tumour, as we are led to suppose in this case that the tumour destroys vision directly by abolition of these ganglia themselves, and not by abolition of some neighbouring or connected part. We have certainly found amaurosis commoner as the tumours approached these bodies, and we have now to investigate the effect of tumours actually in them.

Obscure as the matter yet remains, we do nevertheless attribute visual perception to the corpora quadrigemina, if we make any assertion on the subject at all. Comparative anatomy and experimental physiology seem to point to the quadrigeminal bodies as optic ganglia, if not the only ones. The functions of the geniculate bodies, closely intimate as they are with the optic nerves, seem as yet unknown⁴², nor have we determined the functional relations of the little nucleus in the thalamus which has been so well demonstrated

⁴² It seems probable that they are a medium of communication between the nerves of vision and the hemisphere or brain proper, while the nucleus in the thalamus may coordinate visual with general sensation.

by Dr. Broadbent. These associated ganglia must be concerned with vision, and the comparatively small number of fibres which do reach the quadrigeminal bodies may possibly be only the fibres which co-ordinate the movements of the iris with the impressions upon the optic nerve. After all, however, there does seem to be a certain constancy in the result of early and complete blindness in cases of destruction of the quadrigeminal bodies which we have not found, or have not proved, in the lesions of any other centre. In destruction of the quadrigeminal bodies with amaurosis, we find, moreover, that the early and remarkable dilatation of the pupil also occurs, which is admitted to be due to an interference with their functions in such states as hydrocephalus, and which suggests to us that not only this but the blindness also is due to such interference. I lay the more stress upon this point, as in the two cases of quadrigeminal tumours recorded by Ladame, and in which amaurosis is not mentioned, dilatation of the pupil was also absent. In four other cases collected by myself⁴³, on the other hand, and in which amaurosis was present, dilatation of the pupils is distinctly mentioned as an early and remarkable occurrence. This fact makes me unwilling to accept Ladame's cases as evidence against the connection of amaurosis with lesion of the quadrigemina. The cases may be exceptional, and the mischief did not perhaps destroy the ganglia, though in one case it is certainly reported that the bodies were wholly transformed into tubercles. Whether, in case of destruction of the quadrigemina, other ganglia may still keep up vision, must no doubt remain an open question, but it must be remembered that both Ladame's cases occurred in small children (one a year and three months old, and the other three years old), and that no ophthalmoscopic examination was made in either. Jobert de Lamballe seems to have given special

⁴³ There is a case of tumour of the corpora quadrigemina with early and complete blindness recorded by Dr. Cayley in the sixteenth volume of the 'Pathological Transactions.' I have not included it, however, as it was complicated with hydrocephalus.

attention to amaurosis consecutive to alterations of the corpora quadrigemina, in his '*Études du Système Nerveux.*' I have not had the opportunity of consulting this work, but his observations are quoted by several writers. He recounts several very interesting cases of blindness caused by destruction of these bodies, and in one curious case he discovered a tumour compressing the left corpora more than the right. In this way he explains what was observed during life, namely, that the right eye became blind before the left; which is also in accordance with the conclusions of Flourens. The pupils are said in his cases to have been widely dilated at an early stage of the amaurosis. In two of my four cases I must admit that other parts were affected as well as the quadrigemina. In the first, recorded by Wagner⁴⁴, a new formation of the nature of connective tissue existed in these bodies, but had also involved the pons and posterior fossa. Moreover, the mischief in the eyes was not mere amaurosis, but neuritis⁴⁵. In another case, given by Friedreich, a sarcoma the size of a hen's egg compressed the quadrigeminal bodies, but it originated in the right thalamus. The third case is reported by Rosenthal in his new volume⁴⁶, and I have published an epitome of it in the Appendix (No. 90). In it, again, we are not free from the complication of hydrocephalus, the backward pressure of which upon the corpora quadrigemina always tends to dilate the pupils. The fourth case is one of those of Jobert de Lamballe, to which I have already alluded. Galezowski gives two cases of tumour in which secondary softening of the quadrigemina was found; the tumour in one case was cerebellar, and the reporter was M. Serres; in the other case the tumour was in the posterior cerebral lobe, and was under the observation of M. Renaud

⁴⁴ '*Zehend. Klin. Monatsbl.*' iii. p. 159, (1865). The case is given in the Appendix (No. 89).

⁴⁵ As the amaurosis seems to have been early and complete, there may have been both actions at work. The centres may have been destroyed, and neuritis may also have been propagated along the membranes at the base. I have a strong conviction, however, from the description, that the disks only were inflamed or choked. The sinuses were greatly congested.

⁴⁶ '*Handbuch d. Nervenkrankheiten,*' p. 62.

and himself. These facts are very interesting and important.

On the whole, then, without asserting that obliteration of the quadrigeminal bodies must cause blindness, yet I think that the facts I have adduced, supported as they are by physiological observation and experiment, justify me to some extent in finding in their injury the cause of the amaurosis which also accompanies destruction of parts which lie near them. We should be disposed to explain a case of amaurosis in this way if we found that the blindness was early and soon completed; that the reflex movements of the iris were arrested, the iris being dilated in the periods of destruction, and contracted in stages of irritation; and, thirdly, that loss of vision preceded a progressive atrophy of the optic nerves. Among other symptoms, we should expect palsy of the orbital muscles supplied by the third nerve, and convulsive or paralytic conditions of the limbs, according as the underlying motor strands are more or less involved⁴⁷.

(12) *Tumours of the pons varolii.*

Nothing would be gained by too curious a division of the present subject, so that I omit any separate discussion of tumours of the fourth ventricle and of the pineal gland, which could only be of importance to us in the degree in which such tumours affected regions other than their nominal seat. That tumours of these parts must soon involve the quadrigeminal bodies is obvious, and this is well illustrated by a case recorded in the admirable work of Rilliet and Barthez. An encephaloid tumour was found in the cavity of the fourth ventricle, and during life there was amblyopia, with early and remarkable dilatation of the pupils⁴⁸. It is strange that in a case recorded

⁴⁷ Throughout this volume I have avoided any reasoning from cases not examined after death, but I may be permitted to say that I have a case now under my care which satisfies all these conditions, and another which may be tumour of the corpora quadrigemina, to which I have referred in the section on Tumours of the crura cerebri (page 155).

⁴⁸ *Vide* a curious case of dropsy of the fourth ventricle with amaurosis, recorded in the seventh volume of the 'Pathological Transactions.'

by Virchow⁴⁹, in which the pineal gland was transformed into a bladder the size of a small nut, which pressed upon the corpora quadrigemina, there is no allusion to loss of vision or dilatation of the pupils. There was also a tumour in the left thalamus.

Of clinical with autoptical records of tumours of the pons there is no lack. Ladame gives twenty-six cases, and to these I have quickly been able to add twenty more, four of which were under my own observation almost throughout. Among these forty cases, there are twenty-five in which dimness or loss of vision is recorded; if to these we add on speculation that unknown number in which ophthalmoscopic changes were present but unseen, we shall conclude that defects of the optic nerves are common in tumours of the pons. This we should expect from their nearness both to the centres of vision and to the base of the cranium. Quotations were made in the journals about four years ago from an essay by Da Venezia, entitled '*Sintomatologia dei tumori della protuberanza annulare*' (*Gaz. Med. Lomb.*, No. 15). I have been unable to procure the periodical in question, but I learn that he founds an analysis of symptoms of tumours of the pons upon twenty-six cases, and among disturbances of the parts of vision he notes:—'Convergent strabismus, six cases; divergent strabismus, one case; dilatation of the pupils, four cases; inequality of pupils, two cases; amblyopia and amaurosis, ten cases⁵⁰.' Hydrocephalus in all probability is but a rare cause of amaurosis in tumour of the pons, as I find it recorded in a small proportion only of the cases before me. The nearness of the Sylvian aqueduct would have led us to expect the result of hydrocephalus to be more frequent, and the omission of it in some records may be due to carelessness. It was not present in any degree in my four

⁴⁹ '*Krank. Geschwülste*,' ii. 658, note.

⁵⁰ Many tumours which are really of the pons are nevertheless, so far as position goes, tumours of the posterior fossa also. See, for instance, an interesting case in the '*Pathological Transactions*' (vol. v. p. 26), recorded by Dr. Ogle, in which there was defect of vision. I have called those tumours of the base, however, which originated there, and they are more likely to be attended with meningitis.

cases; but, on the other hand, it was present in a case attended with amaurosis which is recorded by Virchow (*loc. cit.* vol. ii. p. 666, note). The effusion was considerable, flattening the brain; the pons was broader and higher than normal, and contained a tubercular tumour larger than a walnut. There is a similar case recorded by Rosenthal (*loc. cit.* p. 77). I have not been able to satisfy myself concerning any conclusions to be drawn from the site of the tumour in the pons, but I presume that the deeper the tumour the more should we have amaurosis resulting from softening or crushing of the quadrigemina and from hydrocephalus; and that the more superficial the tumour the less likelihood of amaurosis, save in cases of concurrent meningitis. In two of my cases (*vide* 'Path. Trans.' vol. xix. 1868) the tumours were basilar, there was no important meningitis, and the disks were unchanged so long as the corneas remained transparent. In the other two there was tolerably early interference with the disks, and subsequently complete amaurosis, with dilatation of the pupils; the tumours were deep and surrounded by softening which involved the vermiform process on the one hand, and the region of the fourth ventricle, corpora quadrigemina, and (in one case the) crura cerebri on the other. Both these cases occurred in male adults, and the tumours were manifested by many interesting collateral symptoms, upon which I intend to comment at some other time. Meningitis was probably the cause of amaurosis in two cases published by Mr. Salter and Dr. Tyson. Mr. Salter's case is quoted by Ladame from the 'Edinburgh Journal,' vol. xi. p. 270. There were two superficial swellings upon the pons, and there was very considerable exudation upon the arachnoid. There was at first amblyopia in both eyes, and subsequently amaurosis of the right eye. Dr. Tyson's case is recorded in the sixth volume of the 'Philosophical Transactions.' There was a superficial swelling the size of a cherry-stone upon the pons, and chronic meningitis therewith. Amaurosis was among the symptoms. I think we may suspect, therefore, that in cases of tumour of the pons there is no tendency to amaurosis when the tumour is superficial, unless there be meningitis as a middle term,

when we should find in the early stages either neuro-retinitis or ischæmia papillæ: that in deeper tumours, however, the surrounding parts are constantly found to be softened or distorted, such parts being the anterior half of the cerebellum, its crura, the parts near the fourth ventricle including the corpora quadrigemina, and even the crura cerebri. Hydrocephalus, though not a common concurrence, is, however, when it occurs, an efficient cause of amaurosis. In a word, the deeper tumours of the pons, as regards their effects upon vision, resemble tumours of the cerebellum, and of the vermiform process in particular, except in their liability to cause hydrocephalus. Their presence would be betrayed, therefore, rather by progressive atrophy than by choking of the disks. Neuro-retinitis in deep tumours must be rare. The symptoms which would coexist with the ophthalmic signs of tumour of the pons are—direct palsy of the face, and cross palsy and anæsthesia (less constant) of the limbs; or bilateral palsies when the tumour is bilateral, which is less common; headache, frontal or occipital; mental derangements, which are curiously common, and often with depression (Da Venezia found them in thirteen out of twenty-six cases)⁵¹; inflammation of the eyeball; difficulty of swallowing and articulation, and in many cases loss of hearing. Vomiting, apart from dysphagic eructations, is less common, and convulsions are generally absent unless there be some source of irritation, such as meningitis. Da Venezia notes convulsions in but three cases out of twenty-six, and in these three the lesions were not simple ones. The state of the pupils, of which much has been said in lesions of the pons, is inconstant, at any rate in cases of tumour. They seem (amaurotic conditions apart) frequently to have been normal, and, if contracted in a few cases, they have been dilated in as many or more.

(13) *Tumours of the medulla oblongata.*

The effects of tumour in this region are not ascertained, as

⁵¹ It is perhaps not wholly foreign to my purpose to remind the reader that the pons, according to recent researches, seems to be the centre of reaction between emotions from the hemispheres and sensations from the exterior.

the cases seem to be rare. Ladame has only collected nine cases, and I know of no more cases with autopsies which will help us. It is unlikely that tumours of the bulb are attended with amaurosis unless they are complicated with sclerosis or meningitis, or are large enough to involve the encephalic centres above. The occurrence of amaurosis in five out of Ladame's nine cases, seems at first to point to a contrary conclusion; but when we look into these cases, we find that in the first of them there was a large tumour filling the left posterior fossa, and hydrocephalus; in the second a tumour, as large as a chestnut, was placed under the tentorium, and compressed also the pons and the cerebellum; the third and fourth were like unto it; and in the fifth there was also a tumour of the cerebellum, and hydrocephalus⁵². In none of these cases can we pretend that the amaurosis was a result of the tumour in the bulb itself. I think, then, we should only look for ophthalmoscopic signs in cases of tumour of the medulla oblongata when there is coexistent meningitis. I have not met with an autopsy in such a case, however, and merely give this conclusion for what it is worth. As to other symptoms, paralysis of course occurs, but less uniformly than we should suppose; convulsions, on the other hand, seem common; disordered sensations seem to occur irregularly, but are seldom absent. Other changes far more dangerous to life also follow injuries of the medulla, such as neuro-paralytic hyperæmia of the lungs. Hiccough, vomiting, and disordered bowels are likely to occur in such cases, though we do not find many facts at our service to illustrate these points.

May it not be that the medulla is of too vital an importance to the organism to bear much morbid change, and that persons affected with the incipient forms of such change die before the mischief grows to the size of a tumour? The pons varolii, which presides over functions of less immediate value to life, holds out longer, and as its changes are gradually propagated

⁵² In this case the ophthalmoscope was used, and progressive atrophy ascertained. The corpora quadrigemina were crushed by the dropsy, and the optic nerves were very œdematous.

downwards we perceive many symptoms—symptoms among those which I have recorded as belonging to disease of the pons, but which really are evidence of disturbance of the medulla.

(14) *Tumours of the anterior fossa.*

As intracranial tumours approach the base of the skull, we find amaurosis a far more constant symptom,—a symptom, indeed, upon which, in the case of tumours of the anterior and middle fossæ, we may almost count. It was by tumours of the base that we were first taught the importance of medical ophthalmoscopy, and these are the cases which formed the basis of the earliest observations. A tumour of the middle fossa can hardly avoid the optic nerves, and tumours of the anterior fossa must involve them in a large proportion of cases. Tumours of the base set up optic changes in three ways. First, and most commonly, by direct injury to the nerve, severing it from its connections and entailing its atrophy; secondly, by retarding the reflux of blood and choking the disks; thirdly, by irritation of the connective elements of the nerves, with consequent neuro-retinitis. As regards the latter event, I repeat once more that meningitis in all probability is the middle term to which, rather than to the morbid growth, the neuro-retinitis is directly due. Many writers seem to think that it is only necessary to call a tumour a ‘foreign body’ in order to explain neuro-retinitis at once, having the notion, apparently, that ‘foreign bodies’ are fidgetty masses which annoy all the structures with which they come in contact. My experience is, that many foreign bodies get on very comfortably with neighbouring structures, and not infrequently establish tranquil relations of adhesion with them; in such cases the optic nerves are often squeezed quietly to death. In other cases the membranes are inflamed in a way which does not seem to me very easy to explain. In rare cases it may be that a tumour excites, by continuity in the connective tissue, its own process of active nuclear proliferation; but I fancy that the commoner process is the establishment of minute and miliary rents in

the tissue, these rents becoming, each one of them, a centre of inadequate repair.

The anterior fossa or fossæ of the cranium are limited backwards by the smaller wings of the sphenoid, and they lodge the anterior lobes of the brain. Disease of the nose or orbits tends to invade this region, and to set up meningitis or thrombosis of the encephalic sinuses with the optic signs I have described, or to give rise to collections under or about the membranes which are practically tumours. Aneurisms of the anterior cerebral arteries generally belong to this region (*vide* Case No. 80, Appendix), and may cause amaurosis. If so, the amaurosis is of one eye only, one nerve being crushed before the chiasma; in many cases, however, encephalic aneurisms give rise to few or no symptoms until shortly before death. Exostoses and spiculæ not uncommonly take their origin from this part of the cranium, and may or may not wound or crush one or both optic nerves. Caries, again, with the meningitis, abscesses, thrombosis, and kindred accumulations to which it gives rise, exercises ill effects upon the optic nerve, and caries soon reaches the anterior fossæ, if any such mischief be at work below the orbital plates. Tumours of the anterior fossæ, again, are often related in origin to the periosteal dura mater, and by destroying the nutrition of the orbital plates, or crushing them, they encroach upon the orbits themselves. In these cases there is more or less thrusting of the globes outward, which facilitates the diagnosis. A very small depression of the orbital plate will cause a manifest protrusion of the eye, as this deformity, even when slight in degree, soon strikes the observer. In an autopsy upon a case of my own, in which there was, what seemed to us to be, considerable exophthalmos, we found the orbital plate but slightly depressed; the depression, however, had taken place over about two-thirds of the area of the plate, making up in extent for its shallowness. This patient had no difficulty in closing the affected eye. The tumour was fibrous, and involved both optic nerves; the amaurosis was complete.

The perforating tumours, such as encephaloid or 'fungoid'

tumours, frequently make their way into the orbit and push out upon the face, as, on the other hand, they may penetrate from the eye into the head. There are many such instances on record, and they scarcely call for further discussion from the present point of view⁵³. The olfactory lobes, the ethmoid bone, and the nose are often sufferers in cases of tumour of the anterior base; and epistaxis is recorded in some as an early symptom. In a well-known case of Cruveilhier, quoted by Mackenzie⁵⁴ and many other writers, a tumour of the dura mater, seated on the ethmoid bone, and spreading out into both anterior fossæ, destroyed both olfactory lobes and both optic nerves. The patient, whose age and sex are not mentioned, having been long deprived of the sense of smell, and latterly of that of sight, died comatose. It is unnecessary to multiply cases of this kind, which bring their own explanation with them, and offer little difficulty of diagnosis.

(15) *Tumours of the middle fossa.*

It is hard to see how the optic nerves can escape in tumour of the middle fossæ, and as a matter of fact they seldom do. Between the lesser wings of the sphenoid and the superior border of the petrous bone we find the chiasma, the sella turcica, the optic tracts, the third, fourth, and sixth nerves, the ophthalmic division of the fifth, and the cavernous sinus; we have here, moreover, a region very liable to fracture, to caries, to aneurisms, and to tumours. For the effects of fracture, however, I refer the reader to page 111. The pituitary body which lies here has itself a proclivity to disease which, seeing that it lies directly above the chiasma, is very inconvenient for that part. Tumours of the middle fossæ give rise, therefore, to very various symptoms, and affect the disks in sundry ways. If meningitis be present, we shall find ischæmia of the disks or neuro-retinitis; if the tumour directly compress the nerves,

⁵³ Conf. e.g. Gräfe in many places, and especially 'Archiv. f. Ophthal.' vol. i. part 1, p. 417.

⁵⁴ Third edition (1840), p. 869.

we shall find progressive atrophy. These tumours are easy of diagnosis, but an ophthalmoscopic examination may sometimes be the only means of coming to a decision at an early stage. Among tumours of the middle base, the enlargements of the pituitary gland take an important place, for they are not infrequent, and may attain considerable size. As I have said, disease of this kind must crush the chiasma, it will also involve the numerous nerves entering the orbital fissure, and will probably press upon the cavernous sinus. Caries of the sella turcica is not an uncommon event, and brings with it symptoms very like those of tumour. Meningitis of the base interfering with the venous ebb at the cavernous sinus, and perhaps coagulating its contents, thus sets up ischæmia of the disks; or, propagating itself to the sheath and connective trabeculæ of the optic nerves, sets up neuro-retinitis. A case of this kind was under my care four years ago; neuro-retinitis was present during life, and after death I found caries of the saddle, together with meningitis and proliferation within the optic nerve trunk, resembling that shown in the plate. In tumour of the middle base, though both neuro-retinitis and ischæmia may be present, yet primary atrophy is far more common, and is the result of destruction of the trunks or the chiasma by the growth. The pupils will probably be dilated when the atrophy is complete, but will not appear so soon as when the visual centres are compressed by central tumours. I almost think that the absence of all symptomatic change in the fundus would decide me in any given case against the supposition of tumour in the middle fossa, and this I believe is the only locality in which such absence would have a decisive value. For example, an interesting case of this kind came before me two years ago. Mrs. G. was sent to Leeds by Dr. Beaumont of Harrogate, to be under my care. She presented many symptoms of tumour of the base. She suffered from intense paroxysmal headache, vomiting, and diplopia; there was also slight ptosis, and external strabismus of the left eye. These symptoms had lasted about six months. As the disks were absolutely healthy, I told Dr. Beaumont that I felt almost certain there was

no tumour of the middle fossa, and that I thought the absence of neuro-retinitis during so long a time should decide us also against syphilitic meningitis. Nor could we discover any other evidence of syphilis; but still it seemed desirable to give trial to a full and prolonged course of iodide of potassium. This was done with no benefit whatever. As the paralytic symptoms were but slightly pronounced, and varied somewhat with the headache, I ventured to side with the ophthalmoscope, and to pronounce against the supposed presence of any organic disease. Under the care of Dr. Beaumont, and the bold but watchful use of tonic remedies, this lady quite recovered, and has now been in good health for some time. These neuralgic conditions, which may exhibit paresis of both motor and sensitive branches, are not very uncommon; they often mimic meningitis or basilar tumour, but I think they may be distinguished from these latter states by means of the mirror. The cases of tumour or enlargement of the pituitary body recorded by Ladame are fourteen in number, and many more may be quickly added. Strangely enough, Ladame has not included the five cases upon which the well-known essay of M. Rayer was based⁵⁵, nor the similar cases recorded by Morgagni and Bichat. Galezowski refers to other cases again by Chevalier⁵⁶ and by Temputi, an Italian physician⁵⁷. In all these cases sight was affected or abolished, and the optic nerves were found to be crushed. The assertion of Wenzel, that disease of the pituitary body commonly accompanies epilepsy, is not supported by ophthalmoscopic evidence, though it is fair to remember that Wenzel's cases were not cases of tumour. Tumours of the chiasma itself are not very rare; these are sometimes cystic, as in a case quoted by Galezowski, sometimes solid growths, as in the following case put on record by Hjort. A man aged 44 suffered from headache and palsy of the right facial and oculomotorius nerves. The left half of the field of vision was lost on both sides, which corresponded with the discovery after death of a

⁵⁵ 'Archives Générales de Médecine' (1823), t. iii. p. 350.

⁵⁶ 'Journal Universel' (1828), t. xlix. p. 113.

⁵⁷ 'Gaz. Med. Italiana toscana,' 1851. Quoted by Galezowski, *loc. cit.*

tumour the size of a hazel-nut seated upon the right half of the chiasma⁵⁸. The curious precision of the injury in this case might have led to a diagnosis of the origin of the lesion, but of course such tumours cannot as a rule be distinguished by their symptoms from tumours of the pituitary body, nor these again from other small tumours of the middle base. In all such cases we shall find paroxysmal headache in the frontal and temporal regions; ciliary neuralgia; early and effectual injury to the optic nerves, generally of a primarily atrophic character; and palsy of the nerves which traverse the base of the skull, with secondary mischief perhaps of the conjunctiva or eyeball. We shall not find the loss of smell and speech which belong to tumours of the anterior fossæ, on the one hand, nor the loss of hearing and frequent motor palsy which belong to tumours of the posterior fossa on the other. In a very interesting case published by Rosenthal (*loc. cit.* p. 66), melituria accompanied headache and primary atrophy (the eyes were examined by Jäger), the cause being a sarcoma the size of a walnut upon the hypophysis. No mischief was found about the fourth ventricle, but no microscopical examination seems to have been made.

(16) *Tumours of the posterior fossa.*

I have nothing of importance to add here to what has been said in the sections treating of tumours of the pons varolii and of the cerebellum. If the tumour be placed upon the occipital bone, amaurosis is less likely to occur than when it is placed upon the basilar process, or petrous bones. The reasons for this are already explained, being the same as those which explain the occurrence of amaurosis in cases of mischief in the antero-inferior district of the cerebellum. Hydrocephalus, which is not common in tumours of the anterior and middle fossæ, is far more common in tumours of the posterior fossa. It is impossible to say in any given case whether the tumour be placed in the dura mater or

⁵⁸ 'Jahresbericht,' Virchow (1867), ii. p. 497.

in the neighbouring part of the encephalon, nor indeed is it important. A very early and complete interference with either branch of the seventh nerve would be suggestive of the former position, while early paralysis of the limbs would suggest the latter. Neuro-retinitis, being indicative of meningitis, if it occurred early would suggest a tumour of the base; but I have never seen neuro-retinitis in these cases save in syphilitic mischief. As to the frequency of optic signs and their value in diagnosis I have nothing to add to what I have said in previous sections. In my experience the optic signs have always been of late appearance.

Such are the facts concerning the ophthalmic signs of intracranial tumours, so far as we know them. No one admits more readily than I do how few these facts are, and how difficult it is to build up anything upon them. Taking the chapter as a whole, the reader has seen that the occurrence of optic signs is so uncertain that the ophthalmoscope will give no encouragement to the practitioner who takes to it in the hope of making careful thought and quick sense unnecessary. On the other hand, its revelations in many cases are of the greatest value and importance, and in some may even determine a diagnosis. If the subject is to be successfully followed up, it must be by the addition of large numbers of carefully observed autopsies, by the avoidance of those reckless assertions of the certainty of ophthalmoscopic indications in which too many writers have indulged, and by the avoidance also of such smooth phrases as 'the propagation of irritation by a foreign body on the brain along the course of the optic nerve,' and the like. There is little evidence that tumours do propagate 'irritation' in this way, and my own pathological observations seem rather to prove the contrary. I have not argued in the preceding section from any cases, however clear they seemed to be, in which I failed to obtain an autopsy; and I hope that in all future examinations especial attention will be given to the state of the nerve matter surrounding the tumour. Softening and pressure I have found, but not extensive proliferation strolling along devious tracts. There is no mysterious liability in the optic nerves to symp-

tomatic change which is not possessed in a degree by all others; the frequent affection of the optic nerve being due simply to its greater extent in the cranium, to its richness in vascular and connective tissue, and to the size and position of its centres.

IX. *ATHEROMA, SOFTENING AND HÆMORRHAGE.*

In cases of recent hæmorrhage, taken simply, we have but little need for the ophthalmoscope. In a few cases of heavy effusion I have seen a certain amount of venous distension in the retina, either with or without some slight serous effusion also. M. Bouchut gives a number of cases of hæmorrhage, and speaks of such changes in the disk as being very frequent. The cases are by no means satisfactory ones. In many there was no autopsy, and in investigations of the present kind, cases without autopsies are really without value. In Case 74 (*loc. cit.*), indeed, the author actually reverses his proper attitude and diagnoses an apoplexy on the strength of the optic signs. In other cases where an autopsy was made, no account is given of the state of the kidneys. In one or two instances, M. Bouchut's descriptions of the signs in the eyes are very suggestive to me of the first stage of retinitis albuminurica, a condition very likely to appear in such subjects. As an example of the misleading of cases not controlled by autopsy, I may refer to a case published by Dr. Kelly in the '*Lancet*' for Oct. 16, 1869. In this case a clot in the left hemisphere close to the optic thalamus was followed by meningitis, and abundant lymph was found at the base. There was also ventricular dropsy. If the mirror had been used, no doubt changes would have been found in the disks, and might have been quoted as significant of hæmorrhage. In this part of his book, as elsewhere, M. Bouchut has allowed his enthusiasm to get the better of his reason and his accuracy. It would have been at least desirable to state in every case whether albumen was present in the urine or not.

Dr. Hughlings Jackson's opinion on this point coincides so nearly with my own, that I am able to have the advantage

of expressing my own opinion in his words. He says⁵⁹: 'In this case, as in most cases of cerebral hæmorrhage I have seen, there were no abnormal ophthalmoscopic appearances, although the clot was a large one. It has been supposed by some, that large cerebral hæmorrhage leads quickly to acute changes in the optic nerves which, if the patient lives long enough, end in atrophy. Now, whilst admitting that optic neuritis does occur from clot, as it does from other sorts of foreign bodies within the brain, the association is a rare one, and the changes are never seen soon—a few days let us say—after the effusion, never during the stage of insensibility. I have once seen signs of inflammation of the optic nerves in a patient who died in a few days of clot⁶⁰, but this was the exception proving the rule, as the man had had a former attack of cerebral hæmorrhage, and the defect of sight followed six months after this, and twelve months before the fatal seizure. The patient had been under Mr. Hutchinson's care for optic neuritis. Optic neuritis from coarse intracranial disease requires time for its production. Indeed, the proof of this is best supplied by cases of cerebral hæmorrhage, as we know the exact time when the "foreign body" is established in them and not in cases of cerebral tumours. When optic neuritis comes on some time after a clot, we cannot suppose that it has led by "gradual pressure" to interference with the optic nerves. I have once, in a case of extensive meningeal hæmorrhage (which I saw in a patient at the Hospital for the Epileptic and Paralysed, under the care of Dr. Ramskill, who is my colleague there as well as at the London Hospital), seen extreme dilatation of the retinal veins, but in most cases of cerebral hæmorrhage I have found nothing to call abnormal⁶¹.

'In cases of large cerebral hæmorrhage where there is retinal disease with Bright's disease, there are usually (?)

⁵⁹ 'London Hospital Reports,' vol. iv. 1867, p. 345.

⁶⁰ See 'Royal Lond. Ophth. Hosp. Reports,' vol. iv. pt. iv. p. 435.

⁶¹ The pressure of a hæmorrhage is not sufficiently prolonged to bring about the gradual choking of the disks which we see in tumour.

small hæmorrhages about the disks, but so there are in patients similarly unsound when there is no cerebral hæmorrhage, and when the patient does not complain of his sight and can read small print. Yet in the former they are perhaps of larger size and more numerous. I scarcely think it likely that these are due to suddenly added intracranial pressure, but rather that, from some unknown reason, the whole of the cerebral vessels are ready to break, and they break in several places nearly at the same time.'

Such are Dr. Hughlings Jackson's opinions, and such also are the results of my own experience. There are no doubt certain cases where optic neuritis, or more probably atrophy, has followed an encephalic hæmorrhage after the lapse of some or many months, but in these cases the hæmorrhage is but the indirect cause of a change which depends on like changes in the brain mass which surrounds the clot. The clot, or the nerve matter round it, or both, take on a cicatricial change, and so gradually involve the nerve in a cicatricial or sclerotic process. I have never verified this chain of events by an autopsy, but Quaglino says, that in one case amaurosis was found to have been caused by a cicatricial mass replacing a clot which almost entirely replaced the right thalamus. On the left side was found a recent extravasation of blood which was not the cause of the atrophy discovered in the eye. Dr. Hughlings Jackson has also recorded a case in which clot of the left middle lobe, with small extravasations in the tubercular quadrigemina, caused even descending neuritis. This must have been through the means of irritation of the sheath of the nerve.

Hæmorrhage into the retina has been recorded in several cases of encephalic hæmorrhage, but I am not disposed to think that any pressure upon the recurrent vessels could burst the veins of the retina unless these were themselves conspirators. In fact, retinal hæmorrhages, independent of albuminuric retinitis, are not uncommon in old people in whom there is no cerebral hæmorrhage and in whom there may be none. Dr. Sands of New York has published a very interesting case (quoted in '*Edin. Med. Journal*,' 1868, p.

670), in which a hæmorrhage into the sheath of the optic nerve was supposed to be the cause of blindness, neuritis, and atrophy. If such a sequence were verified at an autopsy, we should have gained the certain knowledge that a clot can and does excite inflammation in surrounding nerve tissue where this is rich in connective tissue. I have certainly seen clots excite meningitis; and they might thus indirectly cause amaurosis. A similar case is related most carefully in Pagenstecher's '*Beobachtungen*,' i. 54. Retinal hæmorrhage I have seen several times among Mr. Teale's patients, and I saw it in the case of a patient of my own, who died three years afterwards of apoplexy. Both accidents were said to be determined by a strain; the dimness of vision and retinal hæmorrhage occurred suddenly while he was dragging on a Wellington boot, and the apoplexy occurred apparently in consequence of a muscular effort for another purpose which he had allowed himself to make in spite of warning. The patient was an old gentleman of gouty habit, and with diseased arteries, but not afflicted with kidney disease. Sometimes a small clot is thrown out upon the optic nerves, and compresses them directly, as in Case No. 71 in the Appendix. There are many more cases on record of amaurosis coincident with apoplexy, but the absence or insufficiency of post-mortem notes prevents my making use of them.

The frequent concurrence of albuminuric retinitis and encephalic hæmorrhage in the same person is, however, reason enough to urge us to examine the retina in all cases of encephalic hæmorrhage. The discovery of retinitis or its traces should make us give a far graver opinion than in a case where there was no degeneration of the kidney, and a somewhat graver diagnosis than in cases where albumen in the urine existed without retinitis. It may be the chances of individual experience which lead me to say this, but it has certainly happened to me very often to see retinitis in apoplectics who have been rapidly cut off by a second attack, while others having albuminuria but not retinitis have survived much longer. I may extend this remark further, and say that I

believe a person who has not suffered from apoplexy but who has retinitis is in great danger, though in adding this I am going beyond my present subject. Perhaps I am not exceeding my limits, however, in calling attention to a disorder of vision, not attended with visible change in the eye, which is often to be noticed in patients suffering from a mesocephalic hæmorrhage (see, e.g. Zagorski's case; App. No. 75). This disorder is hemiopia, and depends, no doubt, on the distending pressure of the clot upon one optic tract, for it passes off with the absorption of the effusion. Such patients have satisfactory central vision, but they may be seen to hesitate and grope after objects lying on one side, the dark side of course being the side opposite to the hemiplegia. This functional disorder, for it seems never to advance to atrophy, may remain for some time and be troublesome, but I think it always disappears as the pressure is ultimately removed⁶².

There is another change I have often seen in the eyes of those struck down with hæmorrhage, and that is a certain degree of nerve atrophy. This atrophy is more than mere senile pallor of the disks, it is an actual though incomplete atrophy, and is attended with some degree of loss of vision, though this may not be sufficient to demand serious attention. The disks look white and diminished, and the vessels are fine. I have not been able to ascertain the state of the field of vision in these cases, but this would be an important addition to our knowledge if obtained. I believe that the atrophy is due to atheroma of the encephalic arteries, and thus it may in some degree be recognized as a sign of atheroma and as a forerunner of apoplexy⁶³. If this opinion be confirmed, the sign would have a certain importance, when

⁶² Since this was written, I have met with some observations by Gräfe on the same symptom, and I find from a case of his which I quote in the Appendix (p. 248), that the hemiopia was persistent, and atrophy of the lateral halves of the nerves set in at last, though for five months no such change was detected.

⁶³ It is quite possible that a rigid artery, such as the posterior communicating, might compress the optic nerve and strangulate it. This is a pure hypothesis, however, for I am not aware that such an event has ever been demonstrated.

taken with other things. I have published cases illustrative of this at various times (see, for instance, 'Brit. Med. Journal,' June 20, 1868), but as the confirmation of the rule must depend upon other observers, no good purpose would be served by reprinting them, the proposition itself being too simple to need illustration.

The curious discovery by Charcot of miliary aneurisms in the encephalon, which infest the parts subject to hæmorrhage, and which seem, in many cases at least, to be the immediate cause of the hæmorrhage, suggested to me, and to many others, no doubt, the duty of searching for such aneurisms in the eye. My own search was fruitless enough; but I find from the Bulletin of the Académie des Sciences, in the 'Archives Générales de Médecine' (April, 1870, p. 503), that M. Henri Lionville has been more successful. He appears to have noted the coexistence of miliary aneurisms in the eye and in the encephalon so early as 1868. He now describes the case of a patient, aged 72, who died in the Salpêtrière after several small apoplexies. Innumerable miliary aneurisms were found in the brain, the cerebellum, the pons, and the membranes. There were also aneurisms in both retinas, which latter lesions corresponded to small hæmorrhages in the retinal layers. Examined under the microscope, these aneurisms presented a marked likeness to the changes of the same kind in the encephalic vessels. The report does not say whether these aneurisms were discovered by the mirror; apparently, they were not found until the post-mortem examination.

If we leave recent apoplexies, and turn to ancient ones, we are no longer in want of curious ophthalmic signs. It is a very remarkable and interesting fact that old clots do often give rise to both neuro-retinitis and atrophy, though it is very uncertain how this comes about (vide Cases 72, 73, 74, 76 in the Appendix). Of course it is easy to set neuritis down to 'irritation' propagated by a 'foreign body' lying long in the brain; but we have very little evidence in favour of our accepting this explanation in its only intelligible sense, in the sense that it is a creeping proliferative process. If this be not the explanation I have certainly no other to offer; but in

the only two cases in which I had an opportunity of examining an old clot, I found nothing to support the hypothesis. In one case there were the remains of an old clot in the right posterior cerebral lobe; in the other the remains were in the left corpus striatum. In both cases the clots must originally have been as large as a walnut; and there was in each case a distinct history of an antecedent apoplexy, which in the former case happened a year before, in the latter eighteen months. There was no amaurosis in either case. In both there was a somewhat hardened lump in the brain, faint reddish brown in the centre, and dying off through ochrey and lighter colours at the circumference, where the lumps faded gradually into normal brain tissue. The lumps presented the microscopical characters common to such remains, and in a small belt of brain matter, of perhaps half an inch in diameter, I found an absence of proper tissue, and a presence of connective, degenerative and crystalline elements. I could not, however, in either case find evidence of extensive sclerosis passing to a distance. Still, if secondary sclerosis be not the link between amaurosis and old hæmorrhage occupying parts outside the actual parts of vision, I am at a loss to suggest any other.

Embolism does not give rise to any definite eye mischief, so far as I know, unless it be followed by extensive central softening. The contrary is stated by some writers, and I myself thought it likely that immediately after the accident, when the internal carotid was propelling blood over an area suddenly diminished, there might be some evidence of increased arterial tension, either in dilated vessels or in some degree of effusion. In one or two cases I have noticed some degree of haziness about the borders of the disk and vessels, but nothing that I could with a good conscience call a distinctly morbid state. The phenomena of embolism will, however, be discussed again in a subsequent chapter.

Softening of parts of the encephalon other than the optic centres, tracts, or nerves, without sclerosis, is not attended with any significant changes in the eye. Amaurosis would no doubt result from destruction of the visual centres, or from hæmor-

rhage into them, but I have never been able to satisfy myself of such an occurrence by post-mortem examination. Several such occurrences are, however, on record, the morbid event in the eye being primary atrophy of the disks, following loss of vision. Atrophy also coincides with a certain farther number of cases of central softening; in some it is incomplete, and is probably due, like the softening itself, to atheroma of the vessels; in others, however, it is progressive and unmistakable, when it is probably due to the including of the nervous parts of vision in the central degeneration. Many such cases are recorded by Lancereaux and other writers⁶⁴; but the optic changes occur very capriciously, and have not in their character or mode of occurrence anything sufficiently significant to make it worth our while to discuss them at any greater length (vide Cases 68, 69, 70 in the Appendix). Certainly softening may be present, and often is present in much of the encephalon, without affecting the optic disks in the least, but it can scarcely cut them off from their trophic centre without destroying them. In one very interesting and well-reported case quoted in the Appendix (No. 69), neuro-retinitis undoubtedly accompanied what looked like rapid softening, but may have been encephalitis. I do not know how to explain the neuro-retinitis in this rare case. The optic nerves and centres were not noted after death, nor is the state of the membranes mentioned.

Why in some cases softening involves the nervous parts of vision, and in others does not do so, is hard to say; but it progresses in two different ways at least: first, in arterial regions; secondly, by contiguity, as does gangrene of other organs. Here, however, as elsewhere, when we seek to know where filaments or cells of vision are first involved, we are at a loss for a really sound anatomical basis upon which to work.

⁶⁴ See, for example, a case under Dr. Sibson, in the 'Pathol. Proc.' No. 6, p. 462. Amaurosis accompanied other evidences of widespread intracranial mischief. At the autopsy extensive softening was found without signs of proliferating action, the sheaths of the vessels being, if anything, fatty. The mirror was not in use in 1852; but I am surprised that no mention is made of the state in which the optic nerves and other parts of vision were found after death.

X. CEREBRITIS, ABSCESS, SCLEROSIS.

The chapter on Cerebritis in Reynolds's 'System of Medicine' reminds us of the celebrated chapter on Snakes in Ireland: there is no such thing as cerebritis, the accomplished authors themselves having done the work of St. Patrick. Nothing could be more necessary or more successful than the destructive onslaught of Dr. Reynolds and Dr. Bastian upon the pretensions of all sorts of degenerations in the encephalic mass to call themselves inflammations; but pathologists can scarcely admit that no process whatever exists which can rightly be classed with the inflammations of other organs. The interesting lectures on Analytical Pathology lately published by Dr. Moxon set forth very clearly a result at which we were all arriving, namely, that two chief kinds of 'inflammation' may be classified separately as free inflammations and interstitial inflammations, each class having many peculiar characteristics. Any process, like inflammation, occurring in the encephalic mass, must have the characters of the interstitial kind; it must be like interstitial pneumonia, not like exudative or epithelial pneumonia; it must be like keratitis, not like conjunctivitis; it must consist, that is, in the proliferation of connective elements, not in the proliferation of epithelial or free elements. That the connective element exists scantily and obscurely in the encephalon, makes a great difference in degree between its reaction under irritation and the same reaction in other parts which, like the optic nerve or the lung, are rich in connective elements; but it makes no difference in kind. Interstitial cerebritis is as definite a process as interstitial hepatitis or interstitial nephritis, but it is more difficult to demonstrate, and arises less easily. That the encephalon, as a whole, is certainly not a susceptible organ—not very susceptible, that is, to common irritations—depends mainly upon this poorness of it in the undifferentiated tissues and its richness in tissues which are highly specialized. These highly specialized tissues are as little able to respond by proliferation to simple lesions as a highly specialized

animal to reproduce an amputated claw. Moreover, we must remember that our observations are as yet very incomplete. The number of instances in which the brain tissue around any irritant has been minutely examined, with a view to estimate the secondary proliferation, are as yet very few. For several years I have been hoping to obtain an autopsy in some case of optic neuritis connected with encephalic abscess, and unconnected with meningitis, in order to ascertain whether the neuritis is continuous with a cerebritis propagated from the walls of the abscess, but I have failed. Here again I have to confess to one more of the *hiatus valdè deplendi* which make me doubtful of my right to publish my labours at all. I have twice had the opportunity of examining abscess of the brain from cases in which no examination of the disks had taken place; in neither case did I see the patient alive or dead, nor were the optic nerves forwarded to me. In one case the abscess was encysted, and all evidence of irritation ceased within a short distance of its boundaries; in the second case, the abscess was not encysted, and the evidences of irritation were well marked and widely diffused.

The new fibroid elements and cells penetrated the tissues in many directions, and were best seen in the fresh state. The vessels showed the greatest activity, the sheaths and coats being the seats of an abundant proliferation. No doubt a considerable number of emigrant leucocytes may have been present likewise, and I cannot pretend to be able to distinguish them from young connective corpuscles; but there was an appearance of nuclear activity around the vessels which must have given rise to a considerable proportion of the new elements. As the abscess was approached, the new elements became more evidently what we call pus, and there was a more complete breaking up and liquefaction of the proper tissue of the anterior lobe of the hemisphere in which the abscess occurred. The abscess followed a blow upon the head, but there was no meningitis to be seen. Now, it must be remembered, that a very few elements in this state of high irritation, if they penetrated so far as the optic nerves or tracts, would be competent to set up by continuity a like

process in them; the evidence of irritation becoming then more intense and vigorous as the optic nerves are more vascular and richer in connective tissue. By the penetration of a spark to a more inflammable tissue, the smouldering in the brain is lighted up afresh in the optic nerves. Or if the process be more chronic, if the original focus of irritation be the remains of a blood-clot rather than a lacerated portion of healthy brain, or if it be an old pyæmic settlement, we shall see, instead of a red sclerosis, a white sclerosis, a primary atrophy instead of a neuro-retinitis. Sclerosis occurring in this way may be called propagated sclerosis, to distinguish it from insular sclerosis, in which the disease fixes with apparent caprice upon this part or that, without passing along any perceptible routes. M. Hayem, in a very interesting paper contributed to Brown-Séquard's '*Archives de Physiologie Norm. et Path.*' (vol. i. p. 401), describes the results of some experiments performed by him in Vulpian's laboratory, to ascertain the way in which abscess in the brain is set up. M. Hayem inflicted injuries upon the brain in guinea-pigs, allowed reaction to take place, and then examined the parts after death. M. Hayem found, in cases where a sufficient time had elapsed, that very active proliferation in the neuroglia and in the connective tissue about the vessels took place, and that the formation of cerebral abscess differed in no respect from its formation in other organs. He believes that new blood-vessels also appear in some number. The appearances of cell activity presented a remarkable difference from the like parts breaking down beyond an embolism. In one of his cases he obtained an encysted abscess; and in another, the abscess underwent the caseous transformation.

Abscess, I conceive, may act in three ways upon circum-ambient nervous tissue: it may involve itself in a cyst, and leave the tissues without the cyst unaffected, or nearly so; it may, if not encysted, penetrate into the surrounding tissue for a considerable distance, setting up proliferation in the neuroglia and in the sheaths of the vessels, with liquefaction of the nerve elements; and, thirdly, it may, whether encysted or not, cause those bands of secondary degeneration studied

by Türeck, which are not easily produced by direct experiment, but which are gradually effected by the fell patience of disease⁶⁵. How susceptible the brain is in some states to a distinct and complete inflammation, is proved by the remarkable encephalitis of young children first discovered by Virchow, and since abundantly confirmed by others. In these cases the whole encephalon is in a state of 'neuritis,' the proper tissue being crushed out by an enormous and universal hyperplasia of the neuroglia. I am not aware that any observations of the optic disk have been made in these cases. Still, this shows of what the brain is capable, and how optic neuritis may result from a similar process set up by such an irritant as a bony thorn, an old clot, an abscess, and the like. The observations of Dr. Hughlings Jackson, published in the fourth volume of the 'London Hospital Reports,' certainly prove that 'optic neuritis' is common as a consequence of abscess, whatever the intermediation may be. Two of his cases are especially interesting (Nos. xxv and xxvi). In xxv, an abscess the size of two walnuts lay near the central parts of vision; the mirror revealed 'a state of neuritis.' The disk was obscured by a large patch, and its position known only by the convergence of vessels. The arteries were only visible at the very point of convergence. The veins were large and irregular; patches of blood were scattered about, as if on and smearing a white ground. The diseased patch was about three times the diameter of a normal disk. Both eyes were nearly alike (*loc. cit.* p. 383). Clearly it was a case of neuro-retinitis, with the hæmorrhagic characters I allude to on page 61. At the autopsy there was no meningitis of the base. The parts about the abscess were sent to Dr. Lockhart Clarke, but by a sad misfortune were thrown away. Had they been examined, we should certainly have learnt the answer to the great question—May neuritis descendens be due to a creeping proliferation taking origin in the brain tissue round about a foreign body? We still await the reply.

If the abscess be accompanied, as abscesses secondary to

⁶⁵ Dr. Charlton Bastian has described this condition as consecutive to a hæmorrhage, in the fiftieth volume of the 'Medico-Chirurgical Transactions.'

disease of the nose or ear so commonly are accompanied, by meningitis of the base, then we have a known cause of descending neuritis, a cause which I have repeatedly proved. Then, as I have said (page 85 et seq.), we can demonstrate the creeping of the proliferation from the membranes upon the optic neurilemmata. Traumatic meningitis, so often on the convex surface, can scarcely be the missing link in the cases of abscess with neuritis following a blow.

If an abscess, encysted and not setting up circumambient irritation, press upon the central parts of vision, obliterate the optic nerves, or set up tracts of secondary degeneration, I suppose that atrophy of the disks would be the more or less remote consequence; but I am not aware of any instance in which such an observation has been verified. Nor am I aware of any case in which simple choking of the disks (ischæmia) has resulted from the presence of abscess. An ordinary abscess indeed would scarcely raise the tension of the encephalon to any marked degree, though it certainly might do so if there was a rapid cell growth, or a rapid accumulation of emigrant leucocytes.

It is clear that the irritation or encephalitis set up around 'foreign bodies' is no different in kind from primary sclerosis; 'encephalitis,' 'cerebritis,' 'sclerosis,' 'cirrhosis'—πόλλων ὀνομάτων μόρφῃ μία—may differ in rate, but they are all alike in genesis, or rather in mode. In genesis there is probably a difference between one kind of sclerosis and another. The causes can hardly be the same which give rise to infantile encephalitis, and to insular sclerosis, for example; or, to press matters nearer still, the sclerosis which involves the whole of an optic nerve, and is the result of some distant irritative process closing in upon it, must have a different kind of origin, one would think, from that sclerosis which does not close in upon the nerve in a uniform way, but which exhibits itself upon the nerve in several isolated patches. This latter event belongs to some cause far wider in its action, something which affects the whole cerebro-spinal axis from the optic disks to the cauda equina, and perhaps the organic nervous centres also. In a more diffuse form in general paralysis, in ribbon-like tracts

or bands in locomotor ataxy, and some cases of disease of the anterior columns, or in insular patches, as in sclerosis with palsy, may this fatal change invade the nervous system, its clinical results varying according to its seat rather than according to its nature. All that I may do now is to point out that no part of the nervous system is more obnoxious to this process than are the optic nerves, so that optic atrophy is a common concurrent, and not an uncommon antecedent in these cases. Under General Paralysis I shall show that atrophy of the disks forms an integral part of the disease; and that it forms a part of locomotor ataxy, is familiar to every one.

Finally, in insular sclerosis, or sclerosis with palsy and tremor, we find the same primary atrophy of the disks,—an event which may occur very early in the series of symptoms, or may set in somewhat later. As I have frequently hinted, this liability of the optic nerves may be due to their richness in vessels and in connective tissue; but however this may be, a slow initiation of palsy of the legs, together with atrophy of the optic disks, would lead me strongly to suspect the hidden cause to be insular sclerosis.

To speak even more generally, I have three cases now in my mind, which I do not record because I have had no autopsy in any of them, but in which amaurosis due to atrophy of the optic disks certainly long preceded other manifestations of the three kinds of sclerosis I have mentioned. One man, who attended for some time at Mr. Teale's clinic for amaurosis, and of whom I lost sight, turned up again, a general paralytic, in the West Riding Asylum; the second fell a victim to locomotor ataxy; and the third is now developing symptoms strongly suggestive of palsy with tremor⁶⁶.

I have had no autopsy in any case of my own affected with

⁶⁶ This man (George Airey) now presents symptoms of insular sclerosis which scarcely admit of question. He has been under my care as an out-patient for three years with atrophy of the optic disks, gradual loss of power in the limbs, and tremor on voluntary movement. I do not publish the case at present, as the diagnosis cannot be called quite certain without an autopsy.

insular sclerosis, but the parts from such a case were sent to me about a year ago by my friend Dr. Crichton Browne. The appearances in this case must be described elsewhere; but as regards the optic nerves, I found them in many places reduced to mere connective tissue, with scarcely a trace of nerve fibre. The process was not uniform, but showed harder and whiter patches in places; so that by preserving such parts of the nerves as were not examined, in a small glass tube, I was able to demonstrate to the eye the partial characters of the mischief. I would urge all practitioners, then, whether surgeons or physicians, to avoid speaking too lightly of the consequences of atrophy of the optic disks; if this amaurosis be not as certainly of ill omen as a like discovery of albuminuric retinitis, it nevertheless is but too probable that it may be the first manifestation of some form of sclerosis. Although no wise man would set up unnecessary alarm in the minds of patients or their friends, yet it is his duty to avoid any contrary assurances of safety. We shall do well to remember how such cases may turn out, and to keep such patients under our observation.

In concluding this section, let me warn the reader not to confound with this primary sclerosis a secondary form, which springs up as a result or consequence of nerve wasting. If the fibres, for instance, of the optic nerve waste for any reason, say on account of some disconnection from their centres, then the tendency is seen in connective tissue to replace them. It is fed perhaps on the food set free by the removal of the nerves. However this may be, such hypertrophy is sometimes mistaken for primary sclerosis, and was so mistaken by myself for some time. During this time I was constantly puzzled by observing that in some sections of such optic nerves the nerve fibres wonderfully retained some sort of continuity in spite of great overgrowth of the connective tissue; while in other cases the nerve fibres had fled evidently before the connective tissue could have made any serious encroachments. The former state is seen in primary sclerosis; the latter is seen when the nerve fibres waste from some other cause, and are replaced by an interstitial hypertrophy. Unfortunately I

know of no means of distinguishing the two processes by the mirror. The distinction between atrophies with white, large chalky disks and those with shrunken grey disks does not seem to correspond to any constant or essential difference.

XI. GENERAL PARALYSIS.

As I held that disease of the encephalic vessels was a common cause of atrophy of the optic disks, I entered upon a series of investigations into the state of these parts in general paralysis. The results I obtained were published in the 'Transactions of the Medical and Chirurgical Society' for 1868. I afterwards found that atrophy of the disks in general paralysis had been noticed by Galezowski in one or two cases, by Lancereaux also, and by Von Gräfe, but none of these authors seem to speak from any extended observation. About the same time that I was inquiring into this matter, however, Dr. Westphal, with Gräfe's assistance, was similarly engaged at Berlin, and a copy of his paper on the general progressive paralysis of the insane was kindly sent to me by Dr. Jackson after the publication of my own conclusions⁶⁷. Although I may venture to say that his researches were far less complete than my own, yet it is satisfactory to find that our conclusions are the same. I shall proceed therefore to describe what I myself have seen, without further reference to other writers.

It is a very remarkable fact that in almost all cases of general paralysis there is a tendency to atrophy of the optic nerves. (Vide cases of G. P. in Appendix.) The change seems sometimes to be one of simple atrophy, white from the beginning; in other cases, and perhaps more commonly, the white changes are preceded by a stage of redness, and the whole process then resembles some cases of tobacco amaurosis, and what I have called chronic neuritis⁶⁸. The degenerative process

⁶⁷ This valuable but cumbrously written paper appeared in Griesinger's 'Arch. für Psychiatrie,' &c. The reference to the number is not given upon the separate impression, but the author's date is May, 1867.

⁶⁸ Vide page 64, and the plate of disks in lead poisoning.

begins about the end of the first stage or the beginning of the second, and not infrequently results in complete amaurosis. Probably in most cases of general paralysis great loss of vision might be ascertained in the third stage, if such patients were carefully watched.

The conclusions which I drew in the fifty-first volume of the 'Medico-Chirurgical Transactions' from the careful examination of cases are reprinted in the Appendix, to which the reader is referred for further details.

The pathological characters of the changes in the optic nerves in these cases are a dwindling of the proper nerve structures and an overgrowth of the connective tissue. Now in what relation does this change stand to the brain changes in general paralysis? There are several possible explanations: first, *if* luminous impressions are perceived in the cortical matter of the hemispheres, it might be expected that as the perceptive centres wasted in disease, so the optic nerves would waste from disuse. This, I think, is Lancereaux's view. In wasting of the centres, however, it is not the afferent but the efferent nerves which suffer; and moreover, I have proved by the microscope that if the disease in the optic nerves in general paralysis be not centripetal, at any rate it is not centrifugal, for the optic trunks are often far more diseased than the tracts or corpora quadrigemina. Another explanation, and that by which I was at first tempted into investigation, is, that the degeneration of the optic nerves is due to the atheroma of the vessels. (Vide page 182 of the present volume.) But I think now that the explanation is to be sought rather in the peculiarity of the primary morbid change and in the anatomical characters of the tissues which it invades. How comes this sclerosis or overgrowth of connective tissue about? Is it that some 'irritation,' say of a 'foreign body' or of a 'morbid state of the blood,' excites this tissue, which then grows like thistles on stony ground and chokes the good seed? Or is it that the formative *nisus* is for some reason insufficient to raise material up to the height of nerve tissue, and can create only an inferior product? Or is it, again, that the proper nerve tissue wastes, ceases somehow to

attract and use the blood which passes through the part, and that the connective tissue then battens upon the blood, which it now has to itself? This much it seems safe to say, that the optic nerves are more liable to sclerosis than any other nerves⁶⁹, which indeed we should expect from their vascularity and richness in connective tissue; and that the optic nerves may suffer alone from sclerosis, this remaining the sole nervous lesion, or turning out to be only a forerunner of diffused or patchy sclerosis elsewhere—in the brain, for instance, in the pons, or in the cord. It seems quite certain (*vide* Charcot, 'Gaz. d. Hôpitaux,' 1868, 1869) that, although 'sclerose en plaques' occurs now here and now there, yet that it favours certain parts, e. g. the optic nerves, more especially. The reason of its occurrence in the optic nerves and in other parts of the nervous system must in all likelihood be the same, and depends either upon some disposition of the whole nervous system to a peculiar irritation ('reiz'), or upon some state of the blood, such as lithiasis, acting upon it as a whole.

I am somewhat surprised that in six cases of progressive muscular atrophy, a disease which depends upon patches of sclerosis in the centres, I have not found any degeneration of the optic nerves. In one case, however, external strabismus was an early symptom, and was due perhaps to sclerosis of the third nerve⁷⁰.

⁶⁹ It has been noticed, however, both by myself and others in all the first seven pairs of cranial nerves.

⁷⁰ Vide section on Sclerosis, p. 186.

CHAPTER VI.¹

ON THE OPHTHALMIC SIGNS OF DISEASE OF THE SPINE.

THE curious connection of amaurosis with spinal disease, and especially with locomotor ataxy, has lately attracted much interest. Some observers have endeavoured to explain the concurrence by the hypothesis of an irritation or palsy of mediating vaso-motor nerves; with what truth remains to be seen. In the section on encephalic tumours I have discussed the value of this hypothesis as offered in explanation of the optic nerve changes which accompany such growths within the skull, and I have shown to my own satisfaction that the hypothesis has not the explaining power possessed by certain other hypotheses. As regards the encephalon, however, the admitted obscurity of the causation of optic disorders, and our still greater ignorance of the arrangement and power of any intracranial vasal nerves or centres, aid the reasoner who would connect the two. Here are effects waiting for a cause, and here is an unemployed cause waiting for attributed effects; what could be better than to join the one to the other? In spinal disease, however, this simple and easy way of writing nature down in a book is not so readily applied; for, unfortunately, here we have a few facts, and facts, when they once appear, will grow up like weeds in the trimmest gardens. We do know something about the attachments

¹ A considerable part of this chapter was published in the 'Lancet' of January 15, 1870.

and centres of vasal nerves in the cilio-spinal region and the medulla; we have careful records of many cases in which these nerves or their centres have been severed or irritated by disease or by experimental injury, and we have notes of certain tolerably uniform results which follow such diseases or injuries. Whether among them we find optic neuritis or atrophy of the optic nerves, remains to be seen.

The facts we have to deal with here will be best discussed in the following order:—

Firstly. Do disturbances of the optic nerve and retina commonly follow spinal mischief?

Secondly. If so, then what kind of disturbances are they? And,

Thirdly. What reason or reasons can we assign for their occurrence?

Firstly, then, are the accounts of disturbance of the inner eye secondary to spinal disturbance to be trusted? Of this there is little doubt: it is tolerably certain that disturbance of the optic disk and its neighbourhood is seen to follow disturbance of the spine with sufficient frequency and uniformity to establish the probability of a causal relation between the two events.

I myself examined and tabulated, from this point of view, thirty well-marked cases of *spinal injury*; and in eight of these I found secondary disturbance within the eye. Of these cases, seventeen were severe injuries which proved fatal within a few weeks, and in none of these did changes appear in the eye: the remaining thirteen cases were of chronic spinal disease following accidents of less severity, and it was amongst these thirteen that I discovered the eight cases of concurrent disorder in the eye. Of *acute myelitis* I examined five cases, and in one only did eye disorder supervene. This remarkable case was of very long duration, and was followed by partial recovery; in it disorder of the eye came on many weeks (twelve or thirteen weeks at least), after the subsidence of the acuter symptoms. The myelitis in all these five cases was in the dorsal, or upper lumbar region. Of *chronic degenerations of the cord, exclusive of locomotor ataxy*, I have

records of nine cases. In five of these marked changes in the eye appeared². In *locomotor ataxy* the occurrence of affection of the optic nerve is so well known, that I think it scarcely worth while to sum up my notes of this disease. I wish now to make use of the three following conclusions in particular, which my researches seem to indicate:—

1. That changes at the back of the eye do not infrequently follow spinal disease.

2. That these changes do not become established in the cases which run a short course, but they slowly supervene in the course of weeks or months in more chronic cases.

3. That in spinal disease arising from injury, the higher the seat of the injury the sooner are there changes in the eye. Of this last conclusion I have satisfied myself, after a careful observation of well-marked cases. I have found that the optic changes follow injury to the spine more rapidly if, for example, the injury be in the upper cervical region than if it be in the lower cervical or in the upper dorsal region. One of the best-marked cases of eye disorder with spinal injury that has occurred in my own practice, was in a man who had suffered an injury to the spine in the region of the atlas and third cervical vertebra. The injury was set up by a sudden twist of the head backwards and to the left. In him changes at the back of the eye appeared very quickly and decisively. The patient is still living, but it was clear from the symptoms that the lower cervical and upper dorsal regions were unaffected.

Having seen, then, that there are changes in the eye symptomatic of spinal disease, our second inquiry is—Of what kind are these changes? Confining ourselves to the optic nerve and the retina with their vessels, and omitting all reference to injection of the conjunctiva, or the state of the pupil, what kind of changes are dependent upon disturbance of the spine? I find that they may be well classified under two heads: (1) Simple or primary atrophy of the optic nerve, sometimes accompanied at first by that slight hyperæmia and

² The above numbers refer only to cases which I had watched and noted carefully during a long period, ending January, 1870. A very much larger number of cases have come before me in a more or less passing way.

inactive proliferation which make up the state I have called chronic neuritis. This sort of change I have never found as a result of spinal injuries, but I have often met with it in chronic degeneration of the cord and in locomotor ataxy. (2) A somewhat characteristic hyperæmic change which I have not seen in chronic degeneration or in locomotor ataxy, but in cases of injury to the spine only. The retinal arteries do not dilate, but become indistinguishable; while the veins begin to swell, and become somewhat dark and tortuous. The disk then becomes uniformly reddened, and its borders are lost, the redness or pinkness commencing with increased fine vascularity at the inner border, which thence so invades the white centre and the rest, that the disk is obscured, or its situation known only by the convergence of the vessels³. In many cases, rather than redness, I have observed a delicate pink—pink which sometimes passes into a daffodil colour. In one case in particular—a man at Elland who had been injured in a railway accident and whom I examined in consultation with my friend and colleague, Mr. Teale—this daffodil-colour of the whole field was very curious; no disk was to be distinguished, but the dark vessels stood out in beautiful relief⁴. The other eye presented the more common appearances of hyperæmia and serous effusion, with slight swelling. It is to be remarked, that this state is generally or always of long duration; it passes very slowly up to its full development, and then shows a disposition to end in resolution rather than in atrophy. In those cases which I have been able to watch diligently for many months, the pinkness seems slowly to have receded, leaving an indistinct but not very abnormal disk behind. Sometimes the sight suffers a good deal in these cases, sometimes but little or scarcely at all. I have never seen true neuro-retinitis with active proliferation as a sequel of spinal disease.

³ I need not say that the disk has in reality no borders, but only apparent borders, which are readily blotted out by any loss of transparency in the retina.

⁴ I think these colours are due to the loss of retinal transparency and the blending of its acquired reflections with those of the coats behind it. The state is, I believe, an incomplete or a receding ischæmia papillæ.

The third and most difficult inquiry now remains—What is the reason of the occurrence of these symptomatic changes; what are the processes which, following the changes in the spine and preceding the changes in the eye, link the two events together in the chain of causation? One answer has been lately offered to this question by a distinguished physiologist, Mr. Wharton Jones. His argument is, that when the cord is injured, the sympathetic nerve or its origins are involved; and that, as the sympathetic nerves govern blood-vessels, and blood-vessels govern nutrition, therefore the changes in the nutrition of the eye are due to irritation of the sympathetic, which cuts off arterial blood from the optic nerve, or to the palsy of it, which deluges the nerve with blood. Mr. Jones, indeed, speaks as if dilatation of the arteries at and about the disk were a matter of direct observation. I must say, after some experience with the ophthalmoscope in cerebro-spinal diseases, that this phenomenon has hitherto escaped me. A really satisfactory explanation of the concurrence of spinal and ophthalmic disorders cannot, in truth, be given, until a far greater number of observers have been at work, and until careful autopsies have been made in such cases, with minute examination of the nervous tracts and centres. Meanwhile, no doubt, we must deal more or less in conjecture. The objections to the sympathetic-nerve theory, however, are manifold. The theory is not a new one, and in the chapter on the ophthalmoscopic symptoms of intracranial tumours (p. 119 et seq.), I have already pointed out objections to it which seem fatal. Certain changes said to occur in the fundus in Graves' disease are sometimes called in evidence to show that a diseased sympathetic causes retinal disorder. It is not yet proved that disease of the cervical sympathetic is the secret of Graves' disease; and were it so, the changes in the retina have yet to be determined. I will quote one record of such so-called retinal changes, from an eminent writer who shall be nameless: 'The retinas were observed to be of a bright red colour from injection of the vessels; also on each side of the optic papilla, pigment was deposited in semilunar masses almost black in hue.' This description is absurd, and yet it was not

only pressed into use as an argument in favour of sympathetic nerve causation in exophthalmos, but it was also adduced as a parallel to like pigmentary changes in disease of the suprarenal capsules, which therefore must again be of sympathetic origin !

In the first place, to call up the sympathetic system is to call up too potent an agency for the pressing difficulty. Are we to suppose that the irritated sympathetic causes the destruction of all connected parts ; or that it starves the optic nerve by preference, while it leaves all other parts in its district unaffected ? Or can a palsied sympathetic be the ruin of the optic disk, when its effects are unseen in the pupil, unseen in the conjunctiva, unseen in the ear and cheek ? On the other hand, it is a matter of verified observation, in numerous cases in which there have been most obvious signs of a palsied sympathetic in the ear, face, and outer eye, that in these very cases the back of the eye has been found unchanged. Such is the teaching of Dr. William Ogle's case, read before the Medical and Chirurgical Society on the 23rd of March, 1869 ; and I have a case which is equally instructive in another way under my own care at present. In this little boy, 'a blow upon the nape has set up "strumous" mischief in the cervical portion of the spinal column, with consequent palsy of the arms and legs. During the last few weeks, owing, no doubt, to a lateral extension of the mischief, the left sympathetic in the neck has also become involved, and we have the well-known signs in the left face, namely, narrowed palpebral aperture, injected conjunctiva, undilatable pupil, flushed cheek and ear, and temperature of the cheek ranging from 5° to 9° above the right cheek, except during a febrile access, when this difference ceases or is diminished.' Now in this patient the symptoms of concurrent disorder of the optic nerve and retina were observed in both eyes many weeks before the affection of the cervical sympathetic occurred ; the changes in the eye being of the second kind mentioned above, namely, hyperæmia, with serous exudation ; and there has been no change in the left disk, or in either disk, since the affection of the sympathetic.

Dr. Hughlings Jackson has come to conclusions resembling my own, and I shall therefore quote here an important passage of his bearing on the present subject. It is extracted from the 'Medical Times and Gazette' of October 3, 1863. This skilful observer says:—

‘I have examined the retina in a case of wound of the spinal cord, in which there were on the left side symptoms like those following section of the cervical sympathetic, but I could not find the least difference in the size of the vessels or in the colour of the optic disks, both eyes being under the influence of atropine; besides, there was not, nor had there ever been, any defect of sight whatever.

‘I have had under my care a case of neuralgia of one side of the face, with contraction of the pupil and slight ptosis. As there was, as well as the contraction of the pupil, considerable narrowing of the ocular aperture, it looked, so far, like a case of paralysis of the cervical sympathetic; and as there was neuralgia on the same side in most of the branches of the fifth nerve and hyperæsthesia to the touch, and as the least exertion made the patient sweat on the affected side, this opinion seemed confirmed. The affection was, I consider, in its mechanism, so to speak, I do not say in its cause, a sort of miniature herpes zoster. There was the neuralgic pain, but the changes of nutrition were represented only by a slight haste of the natural functions of the skin, and not by the uproar of actual inflammation. There was something more than paralysis of the branches of the sympathetic correlated with the sensitive nerves to the iris and face, as section of the sympathetic does not produce neuralgia, but this paralysis was, I believe, *one* link in the chain. Although the pupil was only one-third the size of the other, there was no defect of sight whatever. I did not look for defects of sight because the pupil was contracted, but because there might be changes in the retina similar to those in the iris. The patient could read small print easily, and could also see well in the distance. He had no dimness of sight, no “clouds,” “colours,” “specks,” &c., and not a trace of intolerance of light. He could bear, he said, to look at the fire without any annoyance. I dilated

the pupil by atropine, and made an ophthalmoscopic examination. I found no signs of vascularity, the optic disks being as nearly alike as possible. If there were any difference, it was that the disk on the affected side was slightly less coloured than the other. This examination was made soon after the pain had begun, but I examined again a few months afterwards on a relapse of the pain; the pupil being still contracted, I again used atropine. The disks were then quite alike in every respect.

‘I should *a priori* have expected to have found some slight defect of sight and corresponding changes in the vascular supply of the optic disk in such a case, and in the case of paralysis of the cervical sympathetic from injury to the spinal cord. The retinal arteries are supplied with sympathetic nerves as well as the iris, but as I could find no change in their calibre, and no alteration of colour in the optic disk, and as there was no loss of sight and no intolerance of light, nor, in fact, any departure from a healthy state of the retina, it has occurred to me that the part of the cerebro-spinal axis which supplies the vessels of the retina (indirectly by the sympathetic) may be different to that which supplies the iris. If this were so, I should endeavour to ascertain if the same holds good as regards the brain, i. e. if it and the retina are supplied by a different region of the cerebro-spinal axis from that which supplies the external parts of the head, iris, skin, outer ear, &c. The meningeal arteries are chiefly branches of the same great arterial trunks that supply the outside of the head, so that possibly they may be under the control of the same part of the sympathetic system as the iris, outer ear, &c.; whilst the retina and auditory expansion, although outside the cranium, receive branches from the arterial trunks which supply the parts inside, and may be under the government of the part of the sympathetic system which supplies the brain.’

Such is Dr. Jackson’s experience. It is to be desired that some skilful anatomist will really look into the question of the effects of the sympathetic fibres and centres, whether in the neck or in the medulla, upon the cerebral circulation.

Donders and Callenfels certainly state that irritation of the sympathetic and its cervical ganglion in the rabbit caused manifest narrowing of the vessels of the pia mater, followed by paralytic distension. Such distension also followed excision of the cervical ganglion. No note was made in these experiments of the state of the vessels of the retina, and my own attempts to investigate the matter have been defeated by my want of skill as an operator⁵.

Let us turn now from mere vascular changes in the fundus to consider atrophy of the nerve.

Atrophy of the optic nerve, with or without chronic neuritis, is very different in its onset and in its course from the injected fundus; it shows a very different process, and it accompanies very different kinds of spinal disease; the presumption therefore is, that one explanation will not serve for the two sets of phenomena. Moreover, the atrophy, sometimes with chronic neuritis, is commonly met with in locomotor ataxy, and in degenerative conditions of the cord like unto it; but the part of the cord affected in these cases is often away from the connections of the cervical sympathetic; and we know, in addition, that while in locomotor ataxy the degeneration destroys the posterior roots, yet it invariably leaves their ganglia whole. Nor are the threads and centres of the sympathetic itself found, as a matter of experience, to be diseased; if we may rely upon the observations of Friedreich and Carré. Duchenne, I have since read (in the *Gaz. Hebdomadaire*, 1864), found atrophy of the fibres and sclerosis of the ganglia of the sympathicus in two well-marked cases of locomotor

⁵ Drs. Riegel and Jolly have lately repeated these inquiries in Von Ruklinghausen's laboratory. They state in the February number of '*Virchow's Archiv*' that the experiments of Callenfels were insufficient, and they refer also to others by Schultz and by Nothnagel. Schultz says (*Zur Lehre v. d. Blutbewegung im innern d. Schädels*, '*St. Petersburg. Med. Zeitsch.*' xi. 1866, s. 122) that on cutting or irritating the cervical sympathetic in rabbits no change followed in the vessels of the pia mater. Nothnagel ('*Virch. Arch.*' xl. s. 203) agreed with this as a rule, but thought that perhaps the vaso-motor nerves of the pia mater might pass by the sympathetic in a few abnormal cases. R. and J. have, however, made a 'large series' of careful experiments in which they have failed to discover the slightest change in the vessels of the pia mater on cutting or irritating the cervical sympathetic.

ataxy. But what do we find when we turn to the symptoms in these two cases? That there was not only myosis, but vascularity, œdema, and overheat of the face and ear likewise. These objections, not only taken together, but also taken singly, are at least considerable, and, in my opinion, are fatal to a belief in the sympathetic nerve as the cause of those secondary disorders of the eye which we are discussing.

It is less easy to undertake to say what are the causes of disorder of the optic nerve and retina in spinal affections; I shall try, however, to find out in what direction the facts themselves seem to lie.

It is clear, first of all, that we have to do with two distinct kinds of consecutive disorder, and it is probable that they arise from distinct causes. Again, these changes are not peculiar to cases of spinal disease, but they are seen in encephalic disorders also; and, in default of evidence to the contrary, we must assume that their causation is identical or similar in the two. Now, this kind of hyperæmia with serous exudation, when occurring in encephalic disorders, is, so far as my experience goes, very commonly associated with meningitis or extended meningeal congestion of the base; while atrophy or chronic neuritis is either not associated with meningitis, or, if associated with it, is clearly due to other causes—in particular, to disease of the encephalic vessels, to degeneration of the optic fibres or centres, to disseminate sclerosis, or to severance of the continuity of the encephalic optic fibres by pressure, local neuritis, and the like. Hence my former supposition, that the two kinds of change have different causes, is supported by my experience of their causation when dependent on encephalic conditions⁶. Again, as I have said, this hyperæmic state seems to be less a destruction of the nerve than a protracted interference with its vascularity, and this state occurs rather with injuries of the spine than with chronic degenerations of the cord. In these latter cases, when any changes occur, they appear not to be of the nature of a transient interference, but of an essential destruction.

⁶ Another important mark of difference is, that the injected fundus is often unaccompanied by contracted pupils.

These facts seem to support the foregoing: injuries to the spine are very commonly followed by meningeal congestion or meningitis of a subacute character, while slow degenerations of the cord itself are either unattended by meningitis, or the meningitis is a mere local thickening not likely to spread.

In default of a series of autopsies, then, we seem to be led towards the conjecture that hyperæmia of the back of the eye, following injury to the spine, is probably dependent upon a greater or less extension of the meningeal and vascular irritation up to the base of the brain. Now, have we any reason to suppose that such irritation or inflammation does creep up into the encephalon? We have: for, setting aside the curious head symptoms such patients often present, here the actual demonstration of autopsy comes to aid us. It is tolerably well known to careful pathologists that encephalic meningitis is not an uncommon accompaniment of spinal meningitis. I am glad to be able to enlist Mr. Wharton Jones on my own side in this, who makes the same statement himself on the authorities of Ollivier and Abercrombie. It is scarcely needful to point out, that if this explanation of an ascending meningitis be the correct one, it accords with the observation, stated above, that, in general, the higher the injury to the spine, the sooner the affection of the vessels of the inner eye.

Finally, we have learnt, from our experience of encephalic diseases, to attribute atrophy of the disks to severance of the optic nerve fibres, to sclerosis in patches, or to travelling degenerations, rather than to meningitis. Very commonly it is due to what, for brevity's sake, we may call Wallerism, from the well-known experiments of Waller upon the travelling degenerations of nervous fibres. Now, as I have said, atrophy of the disks is seen, not in recent injuries of the spine, but in slow degenerations of the cord—in cases, that is, where meningitis is usually absent or inactive; and it is seen most frequently by far in that degeneration of the cord called sclerosis of the posterior columns, which so often occurs in connection with the extension or the dissemination of like patches of the same process elsewhere, and particularly up-

wards. On the other hand, it seems almost certain, from clinical observation and from autopsy, that sclerosis, both of the optic nerve and of other nerves of the base, such as the third⁷, do accompany sclerosis of the posterior columns with a constancy not accounted for by a propagation of the lesion upwards, or by concomitant mischief of a like kind above the cord; moreover, these nerves are often affected so early, that we can scarcely suppose that the degeneration has had time to advance to the corpora quadrigemina, or other parts of vision, even in the subtlest form. Nay, more; sclerosis of the optic nerves would seem to be actually commoner in locomotor ataxy than in disseminate sclerosis, when lumps of hardened nervous tissue are undoubtedly present both in cord and encephalon. In this latter disease, however—disseminate insular or patchy sclerosis—atrophy of the optic nerves is by no means uncommon. Amaurosis, then, in chronic spinal lesion, will probably be explained when the occurrence of the like changes in the cerebro-spinal mass are explained, and not till that time.

There remains much to be done before we can reason on spinal amaurosis and the ophthalmic signs of spinal injury in any adequate way; and were it not that I thought some handling of the few facts we have would lead to further inquiry, and so far do indirect good, I should have deferred any essay thereon for the present, beset as we are with the double difficulty of indicating the general bearings of phenomena whose connection is so obscure; and, on the other hand, of avoiding the temptation to handle the subject more freely than the actual state of our knowledge can justify.

An important attempt has been made by Dr. Hughlings Jackson to throw some further light upon the subject by direct experiment. The results were negative, but are nevertheless so important, that I think they ought to be republished; I therefore place Dr. Jackson's account of his

⁷ In locomotor ataxy we may find disorders of accommodation, palsy of orbital muscles with double vision, and contraction of the pupils in addition to atrophy of the optic nerve and narrowing of the visual field. The atrophy is in some cases preceded by a transient period of more active irritation.

observations as an appendix to this chapter, to which, perhaps, they have more affinity than to any other part of my book. They may, however, be considered also in connection with his observations on sleep, which I republish hereafter.

Dr. Jackson's communication to the 'Medical Times and Gazette' for the 25th July, 1863, is entitled, *An Experimental Inquiry into the Effect of the Application of Ice to the Back of the Neck on the Retinal Circulation*, and proceeds as follows:—

'At the Hospital for the Epileptic and Paralysed I tried the following experiments, in order to see if I could influence the eye,—the size of the pupil, the calibre of the retinal arteries, and coloration of the optic disk,—by applying cold to the back of the neck. It occurred to me to do so on reading Dr. Chapman's communication in the last Number of the "Medical Times and Gazette," entitled, "A New Method of Treating Disease by Controlling the Circulation of the Blood in Different Parts of the Body." The patient on whom I tried it was a girl of fair general health, but who was subject to sudden startings of the whole body, for which she had been admitted. When ice was applied to the back of the neck and upper part of the dorsal spine, I could detect no alteration whatever in the size of the pupil. I confined my attention to one. It varied readily in light and shade. (I tried this experiment on another little girl several times with the same result.) I then examined the fundus with the ophthalmoscope, noting carefully the size of all the vessels, and the degree of coloration of the optic disk. The nurse then applied the ice to the back at the lower cervical and upper dorsal regions, but there was no change whatever; the vessels remained of the same size, and the disk of the same colour. I next examined when the ice was applied to the back of the head, and then to the side of the neck, and I tried the same experiments on a second patient; in both with the same results.

'Now the pupil not being artificially dilated, it was of course not very easy to estimate the size of the vessels and the coloration of the disk. Yet I had it well and steadily under view when the ice was applied and when it was taken away. I, however, dilated one pupil with atropine, and then

saw the disk as plainly almost as the child's face. Had it, to use such expressions, blushed or paled, it would have been readily detected. I looked both before, during, and after the application of the ice. The large vessels did not alter in the least, nor did a small artery, like a hair, that I watched with great care; and, what is of more importance, I did not detect the least change in the colour of the optic disk. The ice was applied to the back of the neck for exactly nineteen minutes. I was very careful to keep the disk steadily under view the moment the ice was suddenly applied and when it was suddenly taken off, and indeed I kept the disk under a steady gaze nearly the whole of the nineteen minutes. Finding no difference on taking away the ice after this long application, it was re-applied almost directly; and then, the disk being under view, the nurse took the ice away, and immediately substituted a flannel wrung out of very hot water. I could find no change.

‘I used the ordinary ophthalmoscope in the above examinations.

‘Next morning I examined the retina again, and this time by the direct method. The disk seemed, of course, of large size; and I had no difficulty in keeping it under view at the moment of suddenly “making contact,” and of suddenly “breaking contact.” I found no change in the size of the arteries or veins, nor in the coloration of the disk.

‘So far, then, I could see no change in the circulation, no change of colour in the disk; but I freely admit that very likely with the greatest care I might be unable to detect some little alteration of colour. Nor would any one, I suppose, who adopts Dr. Chapman's views, believe that the changes would be very marked, so as to be easily appreciated by the eye. Still, these observations may be of some value, if confirmed, in settling that point by experiment,—viz. that there is no perceptible alteration.

‘I tried then to get information from patients in the hospital who were using the ice for purposes of treatment as to any alteration in the function of the eyes. [The ice is applied for half-an-hour, and then hot flannels for half-an-hour, the

patient sitting in a chair so that there could be no defect from position.] One little girl complained that several times a-day her sight was "queer;" but this is a common complaint in epileptics, and this patient's sight did not fail when the ice was applied. Two other patients were not intelligent enough to give me any answers at all. In a fourth, the answers were unsatisfactory. I asked the patient what she felt, and she described the local sensation, and spoke of a pain over the right eye, and, in reply to several questions, said there was no other feeling. But, on asking the leading question, "Is there anything wrong with your sight when the ice is applied?" she said there was a little dimness.

'My object in making these experiments was not so much for the sake of learning the effect of this treatment on the circulation in the eye, as to be able to form some idea of its effect on the cerebral circulation. Dr. Chapman, in the communication referred to, writes (page 60):—"For example, intending to direct a fuller and more equable flow of blood to the brain, I apply ice to the back of the neck and between the scapulæ." If the circulation in the brain were affected by the application of ice to the back of the neck, it is, I think, probable that the branches of the arteriæ centralis retinae would be affected too.'



The plate is taken from a drawing by Mr. Burgess made for Dr. Hughlings Jackson, who kindly placed it at my disposal. The patient was a man aged about twenty-four years, who had tough arteries, a hypertrophied heart, and albuminuria. No dropsy. He suffered from epistaxis, and from fits of different sorts, supposed to depend upon hæmorrhages into the pia mater. He died from 'apoplexy.' Dr. Jackson went to Portsmouth to persuade the friends to consent to an autopsy, but they refused.

CHAPTER VII.

ON THE RETINITIS ASSOCIATED WITH ALBUMINURIA ¹.

THERE are some cachexies or morbid states of the system, such as that of Bright's disease, of syphilis, of certain poisons, and the like, which affect the eye in very remarkable ways.

Those who are satisfied with current medical language are content to say that these affections are due to the prevailing dyscrasia of the blood. In this and the following chapters I shall shortly describe these phenomena, which are most important to the physician. We shall first consider the amaurosis which occurs in connection with albuminuria. Amaurosis was noticed as a symptom of Bright's disease by Addison, Landouzy², Türk³, Mackenzie, and many other writers of the pre-ophthalmoscopic ages, but the first important descriptions of it are of more recent date. Gräfe, Liebreich, and Desmarres were among the first to give really good descriptions of this amaurosis, with the help of the mirror; and Virchow ('Arch. f. Path. Anat.' x. 170), Müller ('Arch. f. Oph.' iv. pp. 41 and 287), and Schweigger ('Arch. Oph.' Bd. v. Abth. ii.) among the first to investigate the corresponding tissues with the microscope. That affections of sight accompany albuminuria is now well known, though they have not, perhaps, been so well described in English books as they deserve.

¹ *Vide* Liebreich's 'Atlas,' plate ix. figs. 1 and 2, also the woodcut prefixed to this chapter.

² *Vide* 'Ann. d'Ocul.' t. xxii. and xxvi. 1849 and 1850.

³ *Vide* 'Zeitschr. der Wiener Aerzte,' 1850.

It has not been laid down with sufficient clearness, for instance, that in Bright's disease we have to deal with two classes of eye disorders. We may see attacks of partial, or even of absolute blindness coinciding with that state which we call uræmic poisoning, but in which we can detect no retinal changes with the mirror; or we may have impairments of sight which advance more gradually, last longer, and are definitely dependent upon certain visible degenerations.

These two modes of blindness were not distinguished until the mirror threw its light upon them, and they are even now, though clearly described by Förster, and subsequently by Gräfe, but too often confounded by good clinicians.

The first form of Bright's blindness may complicate the second form, occurring at intervals during its course. It consists in repeated attacks of dimness, which accompany headache, dulness, or convulsions; it lasts one, two, three, or more days, and then passes off as the other uræmic symptoms disappear.

The blindness rarely amounts to a loss of perception of light, but often of distinction of objects. It generally comes on suddenly in a few minutes or hours, quickly reaches its height, affects both eyes alike, or nearly so, and either comes and goes at short but uncertain intervals, or varies irregularly in its intensity. This form, which may be called Uræmic Amaurosis, may exist alone; may precede the second, or retinitic form; or may complicate the latter. I have seen, and repeatedly examined with the mirror, three cases at least, in which uræmic amaurosis existed in a severe form, and in which retinitis never appeared; I have seen one case in which uræmic amaurosis preceded the retinal form, and afterwards complicated it; and I have seen numbers of cases in which attacks of uræmic amaurosis came on during the progress of Bright's retinitis, and caused more or less transient increments of blindness.

Gräfe (*loc. cit.*) gives a most interesting case in which uræmic amaurosis preceded retinal mischief; and he says, that out of thirty-two cases of amblyopia with albuminuria, he found thirty in which retinal changes were present, while

in two there were no retinal changes, but there were extreme uræmic symptoms.

The importance of distinguishing between these two forms of blindness is not to be measured only by the importance of the retinal changes themselves, but also by the light it may throw upon the causation of albuminuric retinitis. Were albuminuric retinitis a direct result of the uræmic poisoning, we should scarcely expect ever to find marked uræmia causing blindness, and not setting up a trace of retinitis⁴. On the other hand, again, we frequently find retinitis without uræmic symptoms, as I shall state hereafter. In fourteen cases out of thirty of retinitis, Gräfe (*loc. cit.*) had notes of the absence of any history of uræmic symptoms, while in five more no such symptoms were mentioned. Whether uræmic amaurosis depend upon molecular or vascular changes in the retina itself, or whether (as I think more likely) it is a proper cerebral symptom, cannot as yet be decided. The most likely guess is, that it is due to that anæmia of the nervous centres, with or without serous exudation, which we frequently see in death from uræmia.

I carefully examined the eyes, as I have said, in three cases of extreme uræmic amaurosis, without discovering any variation, even in the vessels; and in neither of these cases was there the presence of convulsions to suggest any periodic instability of the cerebral arteries.

The second kind of amblyopia which accompanies renal mischief is the well-known and not uncommon albuminuric retinitis. Few disorders of the eye present changes so definite and so remarkable. The first stage of this disorder, as seen by the mirror, is one—to use Gräfe's language—of 'diffuse infiltration.' At the commencement, we do not see those spots of degeneration which are so characteristic of later stages, but we see a more or less rapid increase of vascularity. The retina

⁴ Some good cases of mere uræmic amaurosis are published by Ebert and Heusch ('Berl. Med. Gesellsch.' 1868). In a case recorded by Selberg in an inaugural dissertation at Berlin, the amaurosis became complete ('vollständige gewesen sind'), lasted thus two days, and then passed off. The retinas were perfectly normal; there were not even apoplexies, full vessels, or oedema. Gräfe examined the eyes.

for a space of from 3''' to 5''' around the disk becomes slightly swollen, and of a grey or purplish red; many fine vesicles burst into view, and the retinal veins dilate unequally, and grow tortuous; while the arteries tend rather to shrink. The disk is invaded also; it becomes dark red, and suffused; its edges grow dim, and soon a grey, filmy exudation of serum breaks out upon it and upon the surrounding retina, casting a veil over them⁵. The mischiefs may not extend beyond this point, and I believe, from my own observations, that in many cases of recent albuminuria, such as the scarlatinal, complete resolution of this state takes place, and recovery follows. Mr. Bader expresses the same opinion in his paper in 'Guy's Hospital Reports.' If resolution does not take place, however, we have something more to look for. This is the appearance of certain whitish spots and of extravasations of blood in the retina. These spots occur in numbers, and would seem to depend upon a coagulation of the filmy extravasation seen upon the more central portions of the retina, so that central vision is soon deteriorated. They seem rarely to occur upon the peripheral portions. The hæmorrhagic spots may and often do precede the white spots in point of time, especially if the congestive stage has been severe; but I do not adhere to the belief encouraged by Virchow⁶, that the white spots are simply decolourized clots. Many of them may be so; all hæmorrhagic spots, indeed, may turn into white or whitish spots; but I am sure that many of the white spots have never been hæmorrhages. I have repeatedly seen fresh white spots upon such retinas after intervals which were insufficient for the blanching of clot, and, indeed, before any hæmorrhages have occurred; I have also seen the white spots increasing in diameter without any further exudations of blood.

In the admirable 'Atlas' of Liebreich we have two excellent delineations of Bright's retina showing the white spots.

⁵ Liebreich says, that a true intraocular neuritis may occur at this stage, and he gives a drawing of this in his 'Atlas,' Tab. 8, fig. 6.

⁶ Desmarres seems to hold this view likewise in his 'Traité des Maladies des Yeux,' vol. iii.

These, though brilliant, are seldom quite white, but rather of an opaque yellowish or bluish-white colour. The hæmorrhages occur in the inner layers, whence they press outwards; but the spots and patches form, chiefly at least, in the granular layers of the retina, and invade the inner from thence. Not infrequently they may be seen to form behind a blood-vessel, so as to thrust it forward; while at other times they may be seen in front of a blood-vessel, or so including it as to conceal more or less of its length. All these latter patches, however, in my experience, have evidently been clots. Whitish streaks again are seen, which run along the course of the vessels, as though formed by exudation from them, and other white radiating streaks are often visible as the remains of blood outpoured between the bundles of nerve fibres. In the inverted image, with a common two-inch lens, the spots seem to range from the size of a millet-seed or less to the size of a hemp-seed or more. Around the yellow spot is to be seen a group or constellation of peculiar stellate spots, differing both in aspect and origin from those already described. These are due to the fatty degeneration of rods of Müller, the terminations of which they are. As the large white spots and patches increase in number and run together, they tend to surround the disk with a zone, or, to use Liebreich's expression, with a rampart⁷, the inner line of which is irregularly circular, or melts into the grey interval and the disk itself, while the outer presents salient angles which correspond to the course of the larger vessels. This is splendidly seen with a binocular instrument, which brings out the prominences. The large patches have a brilliant and, to the unaccustomed observer, almost a startling appearance. These patches surrounding the disk may gradually invade it, its position having probably, however, been long obscured by congestion, exudation, and proliferation.

The region of the hæmorrhages is the same as that of the spots, namely, around the disk and in the region of the macula. They occur in numbers on the course of vessels,

⁷ 'Arch. f. Oph.' Bd. v. Abth. ii. 1859.

often behind them, or in their angles of bifurcation. They may also, as I have said, form streaks, if they soak between the nerve fibres; but they present themselves more often as irregular dirty blotches among the spots. They are seldom large; in the advanced stages, however, they tend to increase in size, and a sudden and copious hæmorrhage may sometimes be seriously dangerous to the sight, or to as much of it as remains. When we have only to do with scattered, defined spots, and small ecchymoses, we may often hope for great improvement; indeed, if we keep the patient some time under observation, we may even see actual recovery. The retinal mischief always attacks both eyes; but it does not seem to advance and recede with the chief malady. In cases of cure, the blood-vessels begin to shrink considerably in size; those which are decayed, shrivel and vanish; others silt up, and others again seem to present that peculiar condensation and thickening of the walls which is called sclerosis, and which differs from that change in the renal arteries which Dr. George Johnson has called 'hypertrophy,' and of which he showed examples at the British Association Meeting at Oxford in 1868. The hæmorrhages whiten as they recede, commencing at their periphery, and so gradually fade away. The white spots in like manner slowly disappear, remaining longest at their favourite part—the region of the macula. The renewed transparency of the retina allows us now to see that the choroid has suffered, for we find yellow patches have formed in this coat likewise, and the hexagonal cells are so injured that the colour has run. Towards the periphery also the vasa vorticosa have become very visible. This patch-change in the choroid and a persistent retinal anæmia may alone remain to tell of the mischief that has passed by.

In speaking thus of the course of Bright's retinitis, I must be understood, of course, to speak of the eyes singly. Though both eyes are always attacked, yet they suffer generally in unequal degrees, or change at different rates. We may often see the congestive stage in one eye accompanied by the disseminate stage in the other, or the disseminate stage in one accompanied by the patchy stage in the other.

The general symptoms connected with Bright's retinitis, if any, are of course those of the kidney disease.

At the outset of the retinitis, however, patients generally complain of headache. In a little boy, shown to me by Mr. Teale, who was suffering from much headache, after convalescence from scarlatina, we found diffuse infiltration in both eyes. A gentleman once consulted me simply for headache, in whose urine I found albumen; and then, my suspicions being aroused, the congestive stage of retinitis also. As regards the special symptoms of blindness, we find that the loss of sight sometimes creeps on almost imperceptibly, or it may set in rather suddenly. The sight seldom fails quickly, however; and in all cases it observes some various intervals of truce, like the intermittent dyspnœa which we also see in Bright's disease⁸.

In some cases, as in one which I recently had under my care, sudden diminutions of visual power are due to plugging of arterial branches: in one case (App. No. 111), two were thus occluded at an interval of about three weeks. Sudden and large hæmorrhages also often disturb vision, which again recovers itself a little as the blood is reabsorbed; finally, waves of amblyopia may pass over the patient as intercurrent attacks of uræmic amaurosis complicate the retinitis. If the central parts of the retina are attacked first, which is perhaps generally the case, then useful vision is lost early and decidedly, the visual field becomes defective, mists obscure the sight, the outlines of objects become confused, far sight is lost, and coloured vision and persistent blots or images may be present. Some districts of the retina always remain unchanged, and these portions may be seen with the mirror, or may be detected by mapping out the field of vision. Græfe states, however, that in one case of this kind he has seen complete blindness; I presume, of both eyes⁹.

⁸ It is said that this affection of central vision sometimes causes strabismus. I never saw this, and am disposed to doubt it, except as a mere transient state.

⁹ Jaccoud remarks, that sight is seldom quite lost in nephritic retinitis, because the general disease does not give sufficient length of life. This is,

The retinitis never precedes nephritic degeneration, as Landouzy supposed; yet, so silently may shrinking of the kidney go on, that enfeeblement of sight may not infrequently be the first symptom which leads the sufferer to a doctor; and the doctor, who begins by examining the eyes for spectacles, ends by discovering interstitial nephritis. Indeed, the retinitis, so far from being a forerunning symptom, would seem from my experience to be an evidence rather of decided or advanced disease, and would lead me to give a very unfavourable general prognosis. As regards the special prognosis, I think that really good vision is seldom recovered, though Gräfe speaks more cheerfully, and records three cases (*loc. cit.* p. 285) of such fortunate ending. This is a point on which an oculist has better opportunities of judging than a physician.

Such are the features of Bright's retina in its general aspect; let us, in the next place, look at the characters of the tissue changes, as seen under the microscope. In this part of the inquiry almost all that has been written has been taken or derived from the papers of Virchow ('Verhandl. Phys. Med. Gesell.' 2 and 3 Hft. and 'V. Arch.' x. 5. 178), followed by Wagner in same ('V. Arch.' xii. p. 218, 1857); of Heinrich Müller ('Arch. f. Oph.' iv. Abth. 2. 1858); of Gräfe and Schweigger (*ibid.* Bd. vi. Abth. 2. 1860); of Liebreich (*ibid.* Bd. v. Abth. 2. 1859, and 'Atlas,' 1863); and of Müller again (in 'Wurzburg. Med. Zeitschr.' I. i. 1860). In this latter paper Müller gives the first careful description of the changes in the choroid; these are also referred to by Schweigger in the same year (1860), but in a later publication. Able chapters founded on these investigations may be found also in the various subsequent treatises on the eye, as in the 'Lehrbuch' of Stellwag v. Carion, and in the treatises of Wecker, Bader, and the English writers upon diseases of the eye. I shall give a careful account of these investigations, as we scarcely know how much light may be thrown by them now and hereafter upon the intimate

I believe, an error. The process seems after a time to reach a term, and to cease its activity, leaving more or less of the retina destroyed. I have seen this certainly in two cases.

pathology of tissue change in general, and of that remarkable change in particular through which in many persons the whole organism passes in later life, and which shows itself most prominently in a certain coincident degeneration of the heart, arteries, and kidneys. I have before me the treatises to which I have referred, and I have had the opportunity of verifying myself in two cases every statement, or almost every statement, which I shall make.

Mischief is found in three parts of the eye chiefly—in the vitreous humour, in the retina, and in the choroid. The changes in the choroid are substantial, but those in the vitreous are probably but an extension of those in the adjoining retina. However, as we picture the eye from before backward, I shall begin with the vitreous humour.

The changes seen in it are of two kinds, the first being a proliferation of the web of the vitreous, the second being the development of certain linear bodies whose origin is unknown. The proliferation is seen in an increase of the cells of the vitreous, the humour, when hardened, seeming turbid on the outer surface, which is near the retina; some of these cells have processes, but others are round, and by the side of some of them may be seen a hyaline drop, which has escaped on dehiscence of the cell; nuclei again are to be seen, multiplying and dividing, and the substance of the vitreous in these parts has a granular aspect. These changes are best seen in the neighbourhood of the more active tracts of mischief in the retina.

The second variety of products is described by Müller as consisting of little rods (*Stäbchen*) of various lengths, the longer ones being often knotted. Schweigger describes them as fine, slightly varicose, unbranched, somewhat curly threads, which from the periphery inwards lessen until we see them merely as points. These little rods are flexible, and not very brittle; they somewhat resemble spindle-shaped spermatozoa, or fat crystals. That they are not fat crystals, however, is quickly shown by their behaviour under reagents. Like the proliferating cells, they seem to be dependent upon the retinal troubles, for they are most numerous near the affected parts of the retina, and diminish in number as we pass inwards.

I think that the inference we must draw from examination of the vitreous is, that the changes seen have nothing characteristic or special about them, and illustrate only the way in which the humour resents simple irritations. The clinical observer must bear in mind, that this turbidity of the vitreous may often modify the appearance of the retina behind it.

The changes in the retina are as special and peculiar as those in the vitreous humour are simple and general. The appearances I am about to describe are seen after hardening the retina in chromic acid solution, and then making sections of its structure. Let us suppose that the first section of the retina is made vertically, and passes through the disk in the axis of the optic nerve. The first object which attracts the attention is the papilla, which is swollen, but generally still retains its central depression. The surrounding retina is also thickened, but falls away to its normal dimensions as it leaves the disk. At a little distance from the disk, however, we come again upon an increase of thickness as we follow the section through the crown or crescent of white patches which surrounds it. As we leave the disk, we see fatty particles, chiefly in the granular layers, though not wholly confined to them; and these increase as we approach the white patches and the circumvallation, thrusting aside the proper elements of the retina. These fatty granules are to be found sparingly in the inner layers, especially about the yellow spot, and Schweigger speaks also of very fine vessels now to be found in this district. The fat granules are never to be seen in any part of Jacob's membrane.

Besides these fat granules, we discover curious little masses or conglomerations of fibrin, which occur in both of the granular layers and in the intergranular. Near and in the circumvallation these fibrin deposits are less common than the fatty: as we approach the disk, however, the reverse is seen; the fatty elements now fail, and fibrin masses, fibrin threads¹⁰, and actual connective tissue, take their place. Near the disk, the connective tissue of the nerve fibre layer is much deve-

¹⁰ It seems not unlikely that some of the threads or striations in the fibrin deposits may be due to the action of the reagents used in hardening.

loped, and the swelling of the disk is due to a great increase of the same element, with a considerable increase of fine vessels. The proper nerve fibres are seen to be compressed by the new growth, and are, no doubt, much injured in this way; though I have myself reason to think that nerve fibres will bear a great deal of this kind of compression without permanent impairment.

Hæmorrhages, of course, are rarely absent, and are of various ages. They occur in different places, but numerous small ones are generally observed in the nerve-fibre layer, thrusting from the inner to the outer coats. The vessels themselves are sometimes fat dotted, especially those near the disks; or more frequently we may see them sclerosed after the fashion I shall presently mention: finally, they may seem, as to me they seemed for the most part, to be healthy. I have not found any hypertrophy of the muscular layer of the arterioles, nor is it mentioned by any one of the numerous observers which I have quoted. The exact mechanism of the hæmorrhages is therefore not quite clear to me, for fresh hæmorrhages may be seen as streaks in the nerve layer, or as clots in the intergranular layer, without any evident connection either with a fatty or a sclerosed vessel, but nevertheless large enough to crush the outer, or rod and cone layers. All this may be seen with great ease, for in this affection we find, as Müller observes, that the elements show a remarkable '*Isolirbarkeit*,' or separability (if I may coin such a word), which facilitates the distinction of parts, and suggests to us that there must be some degree of sclerosis throughout. Even Müller's rods may be picked out almost entire, and their degeneration around the macula lutea readily verified. This degeneration affects all their divisions, the rods being sometimes obviously fatty up to their termination in the limiting membrane.

The change called sclerosis, to which I have referred, is found in albuminuric retinitis in a well-marked and very curious degree. It affects both the blood-vessels and the nerve fibres, the latter being changed in a way that sorely puzzled the earlier observers. The change in the blood-vessels, which

we shall see also in the chorio-capillaris, consists in a thickening of their walls with a compact, strongly refracting matter, which narrows their lumen. It is found chiefly in the smaller branches and the capillaries, the larger vessels showing rather an increase of the adventitia. The nerve fibres thus affected break up, and form nests of bodies, which resemble ganglion cells, or spindle and caudate connective tissue cells, except in this, that they present no nucleus. Sometimes a certain marking occurs in them, which might lead an unwary observer to suppose a nucleus; but neither this, which I am disposed to attribute to the chromic acid, nor the bodies themselves, display the carmine reaction proper either to ganglion cells or to connective tissue corpuscles. Moreover, Müller gives the following convincing reasons in favour of their origin from nerve fibres. They take their rise always in the fibre layer, never in the ganglion layer, although they may by their size invade the latter. In some places, again, undoubted nerve fibres are seen passing between or about them; and, finally, every transition may be detected between these bodies and the nerve fibres. A fibre becomes irregularly thickened, or varicose; the varicosity is then affected with the peculiar sclerotic change which gives it an opalescent brightness, and the thickened and condensed portion is finally separated, so as to form an independent corpuscle, or rather so as to imitate one. These bodies accumulate in nests, which bulge out the layer which covers them; and among them may be seen likewise some fatty granules which may arise from degenerated connective tissue, or more probably from broken-up nerve (Schweigger). Nerve fibres affected in the way described are often called hypertrophied, which is a foolish and misleading term. I must warn the reader not to mistake for these sclerosed fibres the fibres which are simply enlarged from œdema, and which are to be found in almost all cases of swollen papilla, whether this be due to kidney disease, to cerebral tumour, or any cause whatever. The distinction, when once seen, is easy. The fibres, which are simply swollen, lie almost exclusively about the disk; they do not break off into short lengths, but

are more uniformly enlarged; they do not collect in nests; and, finally, they have not that highly refracting quality which distinguishes the sclerosed portions.

Such in sum are the appearances which have been found in the retinitis of albuminuria; and it is not difficult to account for blindness, when we find from the very first an opacity of the vitreous, a fatty degeneration of the granular layers to a degree which cuts off light from Jacob's membrane and the choroid, and an increase of vascular and connective tissue about the disk and in the optic nerve which must always compress its fibres, and which often, as Liebreich well shows (*vide* 'Atlas,' Tab. 8, fig. 6), amounts to an acute neuritis.

Before passing on to describe the changes found in the choroid, it is important for us to know the degrees of constancy which the retinal changes observe. My own observations are too limited to give much help in this part of the inquiry, but the more extensive researches of Lichtenstein¹¹, Virchow, Müller, Schweigger, and others, seem to prove that sclerosis may occur without the fatty degeneration (Lichtenstein); that fatty degeneration may be far advanced with but little sclerosis, and with no papillary oedema and swelling of nerve fibres (Virchow); finally, that the relations in degree between fatty degeneration and hypertrophy of connective tissue are very variable, even in the same eye. My own limited observations lead me to think that the fatty degeneration and the increase of connective tissue are of the same nature; for in some districts, and apparently in the less vascular districts, there is a rapid proliferation of instable cells and nuclei, while in the neighbourhood of the vascular papilla the new elements are more stable, and weave themselves into a tissue. The sclerosis seems to be a distinct event, always distinct as a process, and often distinct in its time of appearance. It seems at first sight to be misnamed, and to be something different from that which occurs in patches in the cerebrospinal mass; certainly, however, it seems in the vessels to have a community of origin with thickening of the adventitia, and it affects the nerve fibres in that form of encephalitis which

¹¹ 'De Amblyopiâ ex morbo Brightii.' Königsberg, 1857.

Virchow has discovered in newly-born children, and which essentially consists in an irritative movement of the neuroglia. It may, therefore, be of a kindred nature with this latter process, though at first sight so different.

The changes in the choroid in albuminuria are, as I have said, independent of those in the retina, though of like nature to them¹². The so-called sclerosis is here most beautifully seen, and bright patches of it in the chorio-capillaris are visible. Virchow described them first¹³, and Müller and Schweigger admirably followed up his researches. In the choroidal stroma we do not find much mischief. Here and there is a sclerosed vessel; in other places, the pigment structure contiguous to a diseased patch in the chorio-capillaris is atrophied or spoilt, and the pigment cells are all, perhaps, a little too adherent. In addition to this, there is generally some evidence of irritation in the swelling and opacity of connective tissue cells. Schweigger states, moreover, that there may be a discoloration of the stroma at points where no sclerosis is found either in it or in the vascular layer. Hæmorrhages are less commonly found in either layer than in the retina. On stripping off the epithelium, the patches of sclerosis in the chorio-capillaris are at once evident by their peculiar brilliancy. The sclerosed vessels have their walls thickened like those in the retina by a homogeneous strongly-refracting substance, which contracts the lumen, and also makes the outer contour unequal and bulbous. In consequence of the narrowing of the lumen, cavities are formed in places from which the blood corpuscles have often escaped by one or more of the numerous communications. Sometimes, however, an ampulla occurs, which contains a clump of blood-cells. These will be found to be varicosities in the course of arterial trunks. The sclerosis seems to arise in districts, and to frequent the chorio-capillaris chiefly; scattered traces of it may sometimes be found in the veins also, and a few branches of the posterior

¹² Galezowski, in a short essay on the retina in albuminuria, in the '*Union Médicale*,' May 27, 1865, says, I suppose by some oversight, '*La choroïde dans toutes les formes de la rétinite albuminurique, reste intacte.*'

¹³ Virchow, '*Verhandl. phys.-med. Gesellschaft Würzburg*,' x. p. 36.

ciliary arteries may not escape. In addition to the closing of the arterioles by means of sclerotic narrowing and varicosity, we find them closed also by the coagulation of their contents. In this way long plugs are formed, which can be teased or pressed out. A few of them contain some blood-corpuscles, and others may contain granules and small cellular or pseudo-cellular bodies, which may be changed leucocytes, or have had origin in the epithelium; many others, however, are quite transparent and homogeneous.

The epithelium of the ciliary arteries was found by Müller to be much proliferated; in the choroid it is generally loosened or lost, and with it numerous young cells suggestive of proliferation may be seen; there are also spindle-cells full of fat drops, which swell and burst. Free granules may always be seen in abundance. A very interesting kind of peripheral embolism may be followed distinctly in the vessels of this region, the minuter contents of the arterioles being driven into the capillaries. In the chorio-capillaris, indeed, this plugging leads to no great consequences, as the communications are so numerous—more numerous than the plugs. Changes of these kinds, including sclerosis, may be seen also in the fine vessels of the stroma, but to a far less degree than in the chorio-capillaris. As the intimate connection of encephalic apoplexy with kidney mischief makes all changes of the blood-vessels of the retina so interesting, it is curious to see that partly in the affected vessels and partly in their neighbourhood are to be seen many irregular yellowish red or brownish masses, which chiefly consist of blood-colouring matters, the affected parts being ochrey and opaque in appearance.

Some of these are evidently due to mere stagnation and change, but in other places they clearly are the result of extravasation, though neither degeneration nor embolism of the vessels may be discoverable. We are thus obliged to suppose that the exudation is due to some change in the blood rather than in its continents.

Let us now try to sum up what is to be seen in the eye of Bright's disease.

First, we discover a remarkable vascularity of the disk, the old vessels being distended, and a multitude of new ones developed. Besides this, and in intimate genetic relation with it, we find a considerable increase of connective elements in the same parts, namely, in the disk, and in that district of the nerve-fibre layer which lies more immediately around it.

These changes, together with the coincident infiltration of serous and coagulable fluids, cause the loss of transparency, and conceal the vessels and those edges of the choroid and sclerotic which we call the margin of the disk. As these changes advance, we see the hypertrophy of the connective tissue extending itself up to the lamina cribrosa, sometimes even into the depth of the optic nerve, and making itself manifest likewise in overgrowth of the adventitia of the vessels. This latter interference with the vessels causes emptiness of the arteries, with consequent venous fulness, and causes, moreover, those hæmorrhages which occur first, no doubt, in vessels which are fatty or sclerosed, but also in those which appear to be healthy. As we leave the belt around the disk, we find a proliferation of the granular layers of a much more unstable kind, the instability being due, perhaps, to the diminution of blood supply. The products therefore turn quickly into fat, and form a crown, a crescent, or a constellation of opaque, white prominences, surrounding the disk at a certain distance. As time goes on, and the vessels are destroyed, those elements which were formed between the circumvallation and the disk may themselves undergo degenerative change, and the white matter thus gradually invade and include the disk itself.

In the degeneration of the rods of Müller in the less vascular region of the macula lutea, we have a peculiar and very interesting example of this kind of instability of connective elements, and of their transformation into fat. Finally, we notice the sclerosis of the nerve-fibres and of the vessels, the latter being distinctly and independently visible in the choroid likewise. How far the sclerosis may be akin to the fatty change, I cannot say; but my own observations have convinced me that the proper hypertrophy and the fatty degeneration of the connective tissue are one process, and

their difference is not *in essentia*, but *in adjecto*. Herein I must venture to differ from Schweigger and Müller, who conceive, because the two processes take rise in distinct places, and observe each their own district, that they must therefore be distinct in the kind of their genesis.

If I am right, their separation into particular districts is to be explained by the anatomical conditions of the several parts.

I now approach the hard question which must follow: What relation do these changes bear to other changes in the organism, the kidneys of which are diseased; and can we include them in any common system of causation with those other changes? The more I have read upon this matter, and the more I have watched these cases, the more difficult the answer to this question seems to be. I cannot pretend to be able to throw any light upon it myself; nothing is revealed to me which has not been revealed to others, and my only excuse for writing this essay at all is, that I may describe the facts conveniently, and set forth the problem more clearly. I am disappointed to find that I can make no such general co-ordination of the phenomena as to give this essay a permanent value.

In the first place, let us ascertain what are those other changes to which the organisms in question submit. As regards the retinal changes themselves, there is no doubt that they are of a very constant character; they occur, moreover, so frequently, they form so intimate a part of the development of kidney disease, and again, they belong so exclusively to it¹⁴, that we cannot hesitate to endow them collectively with the name of a symptom. They cannot be called merely a complication, still less can they be called an accidental coincidence. Some variations, perhaps, may be seen in the retinal disorder; Pagenstecher and Sämisch believe that there is a form of retinitis gravidarum between retinitis albumi-

¹⁴ So it is, at least if we take them in their assembly. But if we look at each component change singly,—hæmorrhages, granular cells, sclerosis, &c.,—we can parallel them with identical processes in other modes of retinitis. Here again we are defeated in seeking a something characteristic.

nurica and retinitis apoplectica, but I do not think the difference can be more than one of degree¹⁵.

Again, I have myself observed two or three cases in which there has been rather a neuro-retinitis than a patchy retinitis in Bright's disease, and Liebreich's plate referred to ('Atlas,' Tab. 8, fig. 6, ed. 1870) would seem to be an example of this. I have attributed this, however, to a supposed œdema or chronic inflammation of the meninges, like that which in the same patients attacks the pericardium or pleura.

Are the kidney changes, on the other hand, as constant as the retinal? Scarcely so. I have been sadly disappointed in the autopsies needed to settle this matter in three crucial examples, which have been lost by the carelessness of others. In one case, quoted by Dr. Dickenson in his excellent work, I did indeed secure the kidneys from a man who died with well-marked albuminuric retinitis, as witnessed by Mr. Teale, Mr. Oglesby, and my old pupil Mr. Aldridge, and who had well-marked symptoms of epithelial nephritis. The kidneys were large, smooth, and fawn-coloured; but the one which was put aside for minute examination was neglected and lost¹⁶.

Another of the two cases was a young man under the care of my colleague, Dr. Chadwick. He was not more than 25 years of age¹⁷, was a precocious beer-drinker, and worked in the wet. He had the history of epithelial nephritis; scanty smoky urine rich in albumen, back pains, early acute anasarca, waxy complexion, &c.; he had also well-marked

¹⁵ 'Klin. Beobacht. a. d. Augenheilanstalt zu Wiesbaden,' Hft. i. 1861, p. 52.

¹⁶ *Vide* 'Med. Times and Gazette,' May 11, 1867.

¹⁷ But I must caution the reader against the supposition that youth alone forbids us to diagnose granular kidney. Many young women have such kidneys as a consequence of child-bearing, and so have other young people. A very interesting case is related by Schmidt and Wagner ('A. f. O.' xv. 253), in which a 'mädchen,' aged 15, had double neuro-retinitis, with granular kidneys and hypertrophy of the heart. I have never seen such a case as this, however. The neuro-retinitis was like that described by Liebreich, and after death the peripapillary thickening was found to be due to increase of the "outer" layers of the retina. On the other hand, they record a case of encephalic tumour without albuminuria, in which the neuro-retinitis was very like that of albuminuria.

retinitis. He had many attacks of convulsion, and ultimately died at home, and was buried before my sentinel managed to inform me of his decease.

The third case was like unto it in every respect. The young man was under my care in the Infirmary for some weeks with every symptom of tubal nephritis, and died, after several rounds of convulsions. He had retinitis. He died deeply comatose, and, unfortunately, on a day when I did not enter the hospital; by a strange caprice of fate, the autopsy was forgotten until too late, and I learnt his death by seeing his coffin removed by his relations.

Such cases are rarely to be met with, for, whatever be the essential nature of the kidney mischief which accompanies retinitis, this much at least is certain, that it must be of long duration. Let me now turn from my own experience to that of others, in order to learn whether in cases of retinitis other than the granular form of kidney disease has been noticed. The nephritis, caused by pregnancy or retro-peritoneal swellings, and that consequent upon heart disease, are presumably 'granular' in all cases; but setting these aside, we undoubtedly find many cases recorded in which the kidney mischief is of a different kind.

Traube and Beckmann have shown that retinitis has occurred in connection with amyloid kidney; in the case published by Traube, in a lecture which I have before me¹⁸, the disease was very chronic, and the kidney shrunken. The change was undoubtedly amyloid, and followed disease of bone with suppuration. The general symptoms, however, were very like those of granular kidney; the left ventricle of the heart was hypertrophied and dilated, and there was high tension of the aortic system. To this point I shall again refer.

Another important case is recorded by Dr. Russell, of Birmingham¹⁹. In this case, retinitis coexisted with symptoms of epithelial nephritis, and an autopsy was obtained.

In answer to my minuter inquiries about the kidneys, Dr.

¹⁸ 'Deutsche Klinik,' No. 7, Feb. 12, 1859. I have been unable to find any accurate reference to Beckmann's cases.

¹⁹ 'British Medical Journal,' Jan. 15, 1870.

Russell wrote to me on January 22nd, 1870, as follows:—
‘I was unfortunately prevented from being present at the post-mortem in the case you write about, but I received the following particulars: “Kidneys somewhat larger and heavier than natural; capsule peels off easily; surface smooth, pale, fawn-coloured. Heart, left ventricle thick, pale, fawn-coloured; cavities of both ventricles dilated.” I am sorry to say that they neglected to weigh any of the organs.’

In default of microscopic examination, this case is still very important evidence that retinitis does not belong to interstitial nephritis alone; it will be noticed, however, that in this case also the heart was ‘thick.’ Another case of importance was mentioned to me by Dr. Hughlings Jackson some time ago. A woman under his care had retinitis albuminurica, which was carefully noted both by himself and Dr. Pagenstecher. She died shortly afterwards, when ‘typical large white kidneys’ were found. The state of the heart was not noted. In searching through medical literature, one finds numerous examples of retinitis with kidney disease, in which autopsies were made; these for the most part, however, are published by ophthalmic writers, and the description of the kidneys is quite useless. In Dr. Bousseau’s treatise on secondary or symptomatic retinitis²⁰, there are many interesting cases, and some which seem to present symptoms of the larger kidneys; but the descriptions of these organs and of the heart after death are quite worthless. Dr. George Johnson, with whom I have more than once discussed this matter, gives me his strong opinion that retinitis may accompany diseased kidneys other than the granular. He, like myself, has failed in securing autopsies. One of his cases, which was examined by Mr. Wells, I give in the Appendix (No. 109); the other, he tells me in a letter, was a shoemaker, aged 25, a private patient: ‘He had general dropsy, and a large amount of albumen; oily cells and casts were very numerous.’ The history of the case was clearly that of a large white kidney. He died from congestion of the lungs. No post-mortem could be obtained. As he complained of loss of

²⁰ ‘Des rétinites secondaires ou symptomatiques.’ Paris, 1868.

vision I sent him to Mr. Wells, who reported, 'the remains of albuminuric retinitis²¹.' Another fact, tending to associate retinitis with epithelial nephritis, is its frequent occurrence after scarlet fever: cases of this consequence are to be found abundantly in the ophthalmic journals and elsewhere²². I have met with two cases in which kidney disease with retinitis followed scarlet fever, but I have no detailed notes of them, as they were seen by me but once in each instance. These considerations lead me to believe that albuminuric retinitis is not essentially connected with the granular kidney; but I do think that, as in Traube's case and many others, the albuminuria must be of prolonged duration in order to give rise to it. The matter now stands thus; that a definite and peculiar kind of retinitis is associated with chronic disease of the kidneys, and chiefly with that most chronic form, the granular.

It would be, no doubt, of great importance to pathology, could we fill up the necessarily intermediate events. Traube and Schweigger, who are followed by many after writers, attribute the retinal mischief directly to the hypertrophied heart which so commonly accompanies granular kidney. I may go so far as to say, that the occurrence of retinitis, during other kinds of chronic nephritis, does not necessarily forbid the introduction of enlargement of the heart as the middle factor; for I hold that this hypertrophy of the left ventricle may occur likewise in these other chronic kidney diseases. I have already drawn attention to its presence in Traube's and Russell's cases, and Dr. Grainger Stewart found it in 39 per cent. of his cases of tubar nephritis. On further analysis, he found it in 12.5 per cent. of the first stage; in 38.5 per cent. of those in the second stage; and in 100 per cent. of the third stage. I have found it myself also very frequently in chronic nephritis of amyloid and epithelial

²¹ Extract from private letter, dated July 29, 1869.

²² A case is indeed recorded of amaurosis with scarlatinal dropsy, which was cured, together with the kidney disease, by the usual means. (Topinard, 'Gaz. des Hôpitaux,' 1861, quoted by Bousseau, *loc. cit.* p. 49.) No ophthalmoscopic examination is recorded, and the case was probably one of the uræmic amaurosis I have distinguished from retinitis.

nature, in many cases, moreover, in which there was no retinitis²³. On the other hand, I have myself seen three cases of albuminuric nephritis, at least, in which there was no enlargement of the heart whatever, nor any undue tension of the arterial system, as in one of the cases I demonstrated by the sphygmograph. Horner has likewise described cases without hypertrophy of the heart.

When I turn, moreover, to the pithy and accurate reports of Pagenstecher²⁴, I find his testimony strongly in the same direction. He enumerates thirteen cases of albuminuric nephritis, and remarks: 'In all the thirteen cases, we could diagnose heart affections in two only.' This proportion is probably much below the average, but it certainly shows that hypertrophy of the left ventricle is no necessary middle term between the eye disease and the kidney disease. I think hæmorrhages, perhaps, may be commoner, when hypertrophy of the left ventricle is present; but I am not at all sure even of this, and hæmorrhages are indeed common in all kinds of retinitis. It is scarcely necessary to add that the retinitis has never been seen to accompany a hypertrophied heart in cases other than renal. It would seem, then, that this hypertrophy and the retinitis are concomitant effects of some other causes, and are not themselves, as Traube supposes, related directly²⁵.

But, we may ask farther, is there a necessary relation between the retinitis and the nephritis at all? Are they necessarily connected as effect and cause, or are they but common results of some other necessary precedents? That the two are related directly seems almost certain. Landouzy

²³ Hypertrophy of the heart seems to occur sometimes, at least, as a simple result of contraction of the kidney by any means. Dr. Conway Evans found it in a case in which contraction of the kidney was caused by plugging of the renal artery. (*Vide* 'Path. Trans.' xvii. p. 173.) It is difficult to say why this should be; if mere local resistance to blood-passage were the cause, we ought to see the hypertrophy under many other circumstances of the same kind in the liver and elsewhere.

²⁴ *Loc. cit.* Hft. iii. p. 80.

²⁵ I have not opened the question whether the so-called hypertrophied ventricle is any stronger than before. My own observations lead me to doubt this, and to believe that the enlargement of the ventricle is rather of a degenerative kind; but to discuss this would be to leave my proper subject.

is the only writer who has spoken of retinitis as preceding the nephritis, and he evidently did so in ignorance of the subtle character of the granular disease. All clinical experience assures us that nephritis of some kind, and especially of a chronic kind, is a necessary antecedent, and we must hold this opinion until an exception arises. Were it otherwise, were there a single case on record of the retinitis preceding the nephritis, as general arterial disease and a disposition to encroachment of the connective tissue in many other places may undoubtedly precede it—for this my case books can prove—then it might reasonably be held that the retinitis was not directly due to the nephritis, but that both were a common expression of one constitutional tendency,—a tendency which might coexist with other chronic forms of nephritic degeneration. But common as arterial degeneration is in advancing life, I have never yet heard of this condition, apart from the granular kidney, being accompanied by the form of retinitis called albuminuric.

Common, too, as are irritating and prolonged states of lithiasis in patients whose kidneys as yet are sound, I am unaware that this state of the blood has ever been seen associated with the retinitis and not associated with granular kidneys. Apparently, lithiasis alone is insufficient to produce it, and apparently the morbid irritation of the connective elements in the waning organism does not reach the retina until it has first invaded the kidney. Is uræmia the middle term between these two local diseases? This supposition involves us in difficulties almost as great as before. If some one or more of the constituents of urine²⁶, when left behind in the blood, can so irritate delicate tissues as to set up mischief such as we see in the eye, we ought to find some relation between the degree of the uræmia and the degree of the retinitis. Biassed as I am in favour of some such hypothesis as this, yet the only fair ground for it is the constant concurrence of kidney mischief, and of mischief of a chronic kind, which keeps up a

²⁶ Uræmia does not mean, as too many writers suppose, urea in the blood, but *urine* in the blood; and this etymological meaning is fortunately more useful than the conventional one.

prolonged irritation. There seems, also, to be some reason to attribute such a quality to uræmic blood, as this certainly precedes irritative changes in other delicate tissues, such as the nervous and serous tissues. Moreover, it will be seen in the following chapter that a peculiar retinitis is found to occur in many cases of leukæmia, a disease which is attended with excess of urea and uric acid in the blood, and in which we find enlargement of the liver and the spleen, which are now understood to have uropoietic functions. The enlargement of the kidneys in leukæmia is probably but a genuine hypertrophy due to excessive demand upon their excretory function.

On the other hand, however, comes this want of any time relations between the uræmia, or the quality of the urine voided, and the eye disease. Cases are numerous, and have occurred in my own practice, where the retinitis preceded all other obtrusive symptoms, and long preceded any symptoms of uræmia, properly so called; while in numbers of other patients uræmia has been threatening, and long at work, without causing a vestige of a cloud in the eye.

The only remaining hypothesis which I can imagine is, that the blood, impoverished by loss of albumen, tends to exude from the vessels. This, however, would only account for the hæmorrhages; but I think it may explain these and the epistaxis likewise. Certainly many hæmorrhages seem to take place from vessels which are not visibly diseased, and retinal hæmorrhages, like epistaxis, being somewhat independent of the irritative movement, do appear more abundantly in the extremer stages of the disease, while the irritative phenomena may predominate in earlier stages. So at least it has been in my experience. There appears even here, however, the difficulty common to this explanation of the hæmorrhages, and of the epistaxis, that in epithelial nephritis, the loss of albumen and of red corpuscles is certainly as great or greater than in the interstitial, and yet in it retinal hæmorrhages and epistaxis are more rare.

I have failed hitherto to find any common characteristics in kidney patients whose retinas do inflame, and in those whose retinas resist. The immediate cause of the retinitis must be

some such common characteristic or condition, but as yet it eludes me, and I am compelled to leave the matter before the reader in this unsatisfactory state. Still less satisfactory, because more important, is the want of proof of any community in change between the retina and the encephalon, or the contrary. The sclerosis of nerve fibres, so like that seen in this and other forms of retinitis, which Virchow and Hayem have found also in the brains of certain children who died in infancy, and which is probably but an evidence of simple irritation, does not occur, so far as my search is worth anything, in the brains of those who die with granular kidney. I have found no distinct evidence of a proliferating neuroglia, nor of any kindred sub-inflammatory action in the nerve matter²⁷. I should say, then, that the retinal changes would reveal to us but little of the attitude of the encephalic tissues, were it not for a remarkable statement of Galezowski, in the number of the '*Union Médicale*' above quoted. He pursued his researches in a case of albuminuric retinitis from the retina upwards into the encephalon, and he found that 'it is not in the retina alone that the microscope reveals material disorders, for we find them also in the chiasma and the optic tracts. In the right quadrigeminal body we also found a few fatty globules and a considerable development of connective tissue.'

This opens up a new and most interesting inquiry whether the retinal changes really are but the outposts of an extensive chronic cerebritis or sclerosis of parts of the encephalon. If so, we may look for such changes in all cases of chronic albuminuria; and it is much to be desired that such search be prosecuted. As I have said, I have examined the brains from such patients more than once, but have failed to find anything more than the familiar senile changes in the arteries, with the consequent and proportionate deterioration of the ill-nourished districts of the nerve masses.

²⁷ Diseased vessels, and simple granular degeneration as a consequence, is all that I found in these encephala.

CHAPTER VIII.

LEUKÆMIC RETINITIS¹.

WHILE disease of the choroid is seldom symptomatic, disease of the retina is seldom idiopathic; and we find another example of this postulate in the retinitis peculiar to leukæmia. Some years ago, on examining the eyes of a leukæmic patient in the Leeds Infirmary, I found a peculiar retinitis, of which, however, I made no notes, and which I turned to no good account, for my experience of diseased retinas was then not sufficient to show me the value of the observation. The only case I have had the opportunity of examining since was in the Manchester Infirmary, when one day in the wards with Dr. Simpson and Dr. Roberts. The results were negative. Of my own knowledge, then, I can give no information concerning this peculiar symptom, but I shall give a summary of the results indicated by the following writers, whose treatises are before me, and to which I shall add such comments as their observations suggest. These writers are Liebreich², Becker³, Leber⁴, Roth⁵, Simon⁶, and Sämisch⁷. The principal appearances, according to Liebreich, are—(1) A pale colour of all the retinal and choroidal vessels, especially of the retinal veins, which, notwithstanding their repletion and

¹ Liebreich, 'Atlas,' Plate x. fig. 3.

² 'Deutsche Klinik,' 1861, No. 50; 'Atlas,' pp. 20, 21.

³ Knapp's 'Archiv.' vol. i. No. i. Becker gives two chromolithographs.

⁴ 'Klinische Monatsblätter f. Augenheilkunde,' Oct. 1869.

⁵ Virchow, 'Arch.' vol. xlix. Pt. 3, Feb. 22, 1870.

⁶ Simon, 'Zur Lehre von der Leukämie,' in 'Centralblatt,' 1868, No. 53.

⁷ Sämisch, 'Retinitis Leucæmica,' in 'Zeh. klin. Monatsbl.' Oct. 1869, No. 168.

tortuosity, have a light pink shade similar to that of the small apoplexies. (2) Paleness of the papilla, striated cloudiness of the retina in its neighbourhood, and irregular spots near the macula lutea. (3) A number of glistening white round spots, which in form and colour are similar to those found in morbus Brightii, but differ from the latter in their peripheral situation, and hence lie beyond the limits of the drawing. Liebreich seems to have met with such appearances more uniformly than some other observers, as we shall see presently. His experience is, however, so large, as to command much respect, as he has described six cases. He does not say whether these six constitute the whole of the cases examined, or whether some farther cases gave negative results.

Seven years after Liebreich's first publication, Professor Becker confirmed his observations (*loc. cit.*), and more recently again (1869), a communication appears from Leber (*loc. cit.*), which he thinks anatomically confirms Becker's opinion, that the round white spots and the striæ which accompany the vessels are produced by exudation of the white corpuscles; but to this we shall return.

Dr. Roth's examination demonstrated likewise a lymphoid infiltration of the walls of the vessels, which may, however, have been due to emigration: his essay is so painstaking, and moreover differs in so many points from previous accounts, that I have thought it well to make long extracts from it.

It commences as follows:—‘On the 27th of July, 1869, an autopsy was made upon R. T., aged 35, who had been under treatment for splenic leukæmia. The case is recorded by Professor Mosler, in the “Berl. Klin. Wochenschr.” 1869, p. 357. The section made by Professor Grohe displayed an extreme leukæmic state of the blood, a great enlargement of the spleen, liver, and kidneys, miliary tuberculosis of the lungs and right pleura, purulent peritonitis, and pachymeningitis interna, with considerable venous hyperæmia of the brain and pia mater. The eyes, which Professor Grohe had the goodness to hand over to me, were removed with the exception of the anterior thirds.

‘Left eye, vitreous, consistent, and clear; retina somewhat turbid for about four mm. round the disk, otherwise transparent. The chief veins very large and tortuous, full of blood, and dirty red; the arteries of medium size and fulness. About the æquator very numerous hæmorrhages, the largest being the size of a pin’s-head; the inner ones rather opaque; the outer (on the side of the choroid) light red. There are an assemblage of like hæmorrhages behind the æquator.

‘The lacunæ of Blessig on the ora serrata are very wide, the size of pins’-heads, and freely anastomosing. Choroid very full of blood, and of a violet-brown hue.

‘The right eye presents the same conditions, except that the hæmorrhages in the æquator are much more numerous than in the left eye, and one vascular branch passing backwards and inwards from the disk is bordered by two whitish lines.

‘The retinal affection was observed during life by Professor Schirmer, who kindly sent me the following: “In the middle of June of this year I examined both eyes of R. T. with the ophthalmoscope, after I had already examined four other leukæmic patients in Mosler’s wards, without discovering anything particular. This time I found in both eyes a marked retinitis like the plate in Liebreich’s Atlas, that is, there were close upon the disks, especially above and below them, greyish exudations, which veiled the tortuous vessels in the respective parts; likewise around the macula lutea were some uniform specks grouped concentrically, and moreover there were a few slight apoplexies near the veins. On the other hand, I failed to see the tint of the fundus described by Liebreich and the bluish veins. The fundus was at most a little brighter than normal. I had no opportunity of making a later examination of the patient.” Disturbance of vision was not then present, nor was it observed subsequently.

‘The left retina was first examined by the microscope. In the fresh state, granule cells of various size and form were conspicuous upon the numerous little vessels at the periphery, especially upon the capillaries. Almost all the vessels also were enormously full, some being equably distended, others varicose. White blood corpuscles were numerous, but on account of

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their unequal distribution could not be accurately estimated. The chief localities for the accumulation of colourless elements were the varicosities of the capillaries. Here red corpuscles were often quite absent; while the white, pressed together, in numbers of twenty or more, until their contours were obliterated, gave, with the closely investing capillary membrane, the deceptive appearance of a gigantic, many-nucleated cell. These accumulations of white cells in leukæmia are not unusual, and have already been described by Virchow and Bennett in the capillaries of the brain and lungs: according to Charlton Bastian ("Brit. Med. Journ." 1860), emboli may thus be formed. In one case the fusion was but apparent, and the masses could be resolved again into common white corpuscles, when the retina had been hardened in Müller's liquid.

'As regards the position of the hæmorrhages, their already mentioned frequency in the peripheric portions was evident in the first instance, and secondly, their more common occurrence in the outer layers of the retina. Even to the naked eye this seemed their probable position, as the hæmorrhages seen from the inner surface appeared rather more opaque than from without. In vertical sections were found little heaps of red corpuscles between the inner granular layer and the external limitary membrane; the inner layers were more rarely the seat of the bleeding. How far fatty degeneration of the walls of the vessels had disposed to hæmorrhage, must be left undetermined; in every case, indeed, extravasations were seen issuing from vessels which did not present any structural change.

'The lacunæ near the ora serrata were considerably dilated, the circulation being, in fact, chiefly interfered with in the peripheral parts. Under the microscope, the smaller gaps were seen to be confined to the inner or outer granule layer, while the larger ones occupied almost the whole thickness of the retina.

'The examination of the fundus within the turbid zone was made upon preparations hardened and coloured with osmium and carmine. As regards the larger vessels, it was to be remarked, that they presented but few free nucleated cells;

some of them showed a thickening of the adventitia, caused by a 2—5 fold layer of pale round cells of 0.008 mm. diameter, for the most part, or sometimes of 0.006—0.01. mm. The large vessels appeared much dilated, thrusting the limitans interna somewhat inwards, and so separating the outer more yielding layers from each other, that only the outermost granules and the limitans externa with the adherent rods passed over the vessels.

‘Hæmorrhages in this district were few, as before said, and here also affected the outer layers of the retina for the most part. Some discrete brown masses of pigment about the disk represented some earlier effusions. The thickness of the retina and of its individual layers, taken all together, was not increased, and tallied pretty nearly with Heinrich Müller’s standard measurements.

‘The supposition that the opalescence of the fundus depended upon cedema found no support. On the contrary, the outer fibre layer in this region was finely granular throughout, and indeed there were granules, which took a brown colour with osmium, enclosed in Müller’s rods, which rods were here very greatly developed, and presented wing-like appendages. In many places the rods were distended in a spindle-shaped fashion, and swollen to the size of 0.025 mm. in length and 0.01 mm. in thickness. Sometimes they presented also a pale, nuclear body of 0.007 mm. in length and 0.005 mm. in breadth. This enlargement and the granular aspect of the rods of Müller were the only changes in the outer fibre layer which would account for the diffuse opacity of the fundus.’

Some defects of the rods and cones were probably due to maceration, and the other layers of the retina, saving a few granule cells in the inner granule layer, presented no changes, —but the optic nerve layer contained several little nests of hypertrophied nerve fibres near the macula lutea. ‘The largest of them was situated 4 mm. to the outer side of the central veins; it was spindle-shaped, 0.3 mm. long, 0.16 mm. thick, and bulged; the limitans interna was thrust somewhat forwards, so that the nerve layer seemed almost three times as thick as elsewhere. Here were a large number of those peculiar bullet

and club-shaped bodies, made known by Zenker and Virchow, which were sometimes pale and homogeneous, sometimes very brilliant, and often contained a nucleoid structure. Their size varied between 0.005—0.078 in length, whilst their greatest thickness rarely surpassed 0.03. In the largest spindle-shaped corpuscle, which was 0.078 in length, the round nucleus within measured 0.02—0.01 in thickness. In the preparation, which was first tinted with osmium and then with carmine, the outer corpuscle appeared of a red brown, and the nucleus of a clear brown, while on simple carmine staining the nucleus took a very intense red. The smaller nests, situated likewise in the neighbourhood of the macula lutea, consisted only of five or six, often of still fewer sclerosed fibres. In one place the boundary of such a fibre was irregularly infiltrated with pale, round, and elliptic nuclei of 0.006—0.011 mm. in size, which presented a great resemblance to the nuclei here developed in the rods of Müller. (Kölliker, "*Gewebelehre*," 5 Aufl. S. 682.)

‘The optic nerve entrance was examined by perpendicular sections without anything abnormal being found; all the vessels here were stuffed with blood; generally speaking, in the capillaries there were many more red corpuscles than in those of the retina. In particular, the interstitial connective tissue and the sheath presented no trace of lymphoid growth; also a few sections of the nerve, made about half an inch behind the disk, were quite normal in character.

‘The principal change in the choroid consisted in the marked hyperæmia, already mentioned, which was equally distributed throughout the chorio-capillaris and the choroid proper up to the vasa vorticosa. In the larger vessels were considerable collections of white blood corpuscles, held together by delicate fibrin threads, probably post-mortem coagulations, while in the capillary vessels the number of red and white corpuscles was about equal. The isolated blood corpuscles without the vessels were, for the most part, singly nucleated, though some contained two or three small nuclei, and measured from 0.008—0.012 mm.

‘In the posterior division of the choroid were some isolated

jelly-like bodies, which existed very numerously in the anterior portion, and were seated upon the elastic lamina. They measured from 0.02—0.18 mm. in diameter, and presented the same characters as depicted by Donders and H. Müller.

‘The right eye was only partially examined, but changes were found in front of the equator as in the left eye. Thus, in the fundus there was the same opacity and hypertrophy of Müller’s fibres, but sclerosed nerve fibres, on the contrary, were not found. The disk presented no anomaly; and the choroid had the same characters as in the other eye, except that within and above the disk was found an irregular infiltration of colourless, round, and club-shaped cells of 0.008—0.012 mm. in size with nuclei of 0.006 mm., or sometimes with several small nuclei. The infiltration extended through the chorio-capillaris and the choroid proper as far as the inner layer of the supra-choroid. In spite of the connective tissue growth, the choroid did not seem in these places to be thickened as the vessels were correspondingly narrowed. The infiltration ceased at the choroidal opening, but its extent outwards was not determined.’

If we sum up shortly the principal results from the foregoing description, they comprise hæmorrhages seated chiefly in the peripheral districts and in the outer layers of the retina, far-gone fatty degeneration of the retinal vessels (especially at the periphery), and well-marked ectasis of the marginal spaces (Blessig); diffuse opacity and hypertrophy of Müller’s rods; thickening of the larger blood-vessels, which in some places have the appearance, even to the naked eye, of a whitish bordering; sclerosis of nerve fibres about the macula lutea in the left eye, and a circumscribed lymphoid proliferation (?) in the choroid of the right eye.

A glance at the existing literature of leukæmic retinitis (*vide* Leber, Zehender’s Monatsbl. 1869, S. 312 et sq., besides the case of Sämisch, *ibid.* S. 305), suffices to show the great variety of changes possible in it; we shall only be enabled to comprehend these in one point of view, when we possess thoroughly complete clinical observations, in addition to anatomical investigations. For instance, there is but one

point in this case of Dr. Roth which agrees with the changes described as characteristic by Leber, and that is the peripheral situation of the hæmorrhages; and even here there is this distinction between them,—that the bleedings took place in the outer layers of the retina from the vessels which originated in the inner granule layer, whilst in his case the hæmorrhages belonged strictly to the inner layers. In a case very recently published by Dr. Reincke again we shall see that hæmorrhages occurred throughout the thickness of the coats.

It is worthy of remark, that in the case of Dr. Roth the hæmorrhages were not of the dull red so often described in leukæmia, but had an intense red colour, toned down only by the covering of the inner layers of the retina; they were proved also under the microscope to consist of red corpuscles closely pressed together.

The discrepancy between the ophthalmoscopic and the anatomical investigations in the present case was very likely due to the considerable interval (about six weeks) that intervened between the two.

On turning now to other evidence, we find that Becker (*loc. cit.*) gives the following description of the appearances in one of his two cases. The patient was a man, aged 32, who had suffered from intermittent fever, and also from syphilis in all its stages. He had latterly splenic leukæmia and amblyopia, especially of the left eye. The fundus of this eye presented interesting changes: it was of an orange yellow, the borders of the disk slightly veiled; the veins pale rose, and very broad; while the arteries were thin, distinct, and pale yellow. At the macula lutea was seen a bright yellowish white spot of about one-fourth the size of the disk; it was surrounded by a red border and some small white specks. This coincided with an almost central scotoma, which diminished as the red border of the patch grew pale. A like yellow prominence appeared while the patient was under observation. It took its rise from a vein at a point about three diameters distant from the disk. The vein was very tortuous, and bordered on both sides with dull yellow stripes. Becker thinks that the yellow patches were nests of migrated white

corpuscles; it is more probable that they were actual hæmorrhages, hæmorrhages having a centre of white corpuscles and a capsule of red corpuscles, as we have seen was demonstrated microscopically in other cases; as, for instance, in the following case of Leber:—Leber found the retina of the fresh eye slightly turbid and beset, in its anterior section especially, with numerous round, white, prominent spots, of one millimetre in size at most, and surrounded by a red areola. Only a few of these were red in the centre, but near them were also some hæmorrhagic spots also. Some vessels were bordered by streaks. The larger whitish spots were seen in the hardened retina to consist of aggregated red and white corpuscles, occupying in a few places the whole thickness of the coat. There were no perceptible changes in the vessels, but some vessels in the disk were seen to be compressed by layers of closely-packed lymph bodies. The lesser collections lay in the fibre layer, and left the granule layer free. The blood in the retinal vessels was full of white corpuscles. Choroid unchanged, but very hyperæmic; in one optic nerve was a very limited fatty degeneration of nerve fibres. Leber is strongly of opinion that the masses in the retina were only hæmorrhagic in small part, and were chiefly to be regarded as minute leukæmic tumours (*kleiner leucämischer Geschwulstheerde*). He bases this opinion upon their rounded form, their prominence, the possession of a red areola, and their frequency in the periphery. These arguments seem to me insufficient. Leber supposes, I presume, that this red areola was an evidence of reaction in the surrounding tissue, but he gives us no microscopic evidence of such reaction. In Dr. Roth's case we had evidence of retinitis, but in this of Leber's no changes are described which can be fairly classed as irritative. And I shall now go on to describe a case recorded by Sämisch (*loc. cit.*), which, when taken with another published by Dr. Reincke, an assistant of Prof. Engel-Reimers of Hamburg, tends rather to show that these spots are merely hæmorrhages. That there is a great tendency to hæmorrhage in leukæmia is familiar to us all, and is borne out as regards nervous tissues by the frequency of encephalic hæmorrhages

in these cases ; not only so, but in the eye itself there is evidently a great tendency to hæmorrhage, as in Simon's case (*loc. cit.*) there was a hæmorrhage upon the sclerotic, and in that recorded by Sämisch, sudden loss of vision in the right eye was caused by a large extravasation within it. In the left eye of this latter case, which had also been somewhat dim of vision, was found some turbidity of the hinder section of the retina, and also apoplexies, and white spots upon it, which white spots lay near the vessels. Not only the hæmorrhages but the white spots also were seen to diminish, and new ones to form. There were in this eye moreover large apoplexies in the equatorial district of the choroid. The following appearances were noted in the eyes, after hardening in Müller's liquid :—

R. eye. Vitreous contained numerous products ('Derivate') of blood corpuscles. There was an atrophied district 9 mm. broad by 4 mm. broad at the lower part of the retina.

L. eye. The inner granule layer of the retina was much thickened in parts where were mulberry-like masses of blood corpuscles. The adventitia of the larger arteries was thickened, the connective framework œdematous near the disk, and rich in nuclei within it. The vessels of the choroid were full of blood in both eyes, and its stroma contained heaps of corpuscles. Here there were slight evidences of irritation. There was also marked choroidal mischief, which Sämisch supposes to play an important part in leukæmic retinitis. There was no choroidal mischief, however, in Leber's case, nor in that of Dr. Reincke⁸. Dr. Reincke, in examining the many encephalic bleedings which he found in his case, saw that in a large number of them blood was effused in rings between the media and the adventitia, and between the intima and the media. In the larger number, however, effusion had actually taken place into the nerve substance, though even in these a streaked disposition along the vessels might often be traced. These streaks, he says, in many places suggested most strongly the notion of an emigration of leucocytes ; but this notion was quickly dispelled by following some of them up to a point where they became continuous with unques-

⁸ Virchow, 'Arch.' vol. li. part 3.

tionable hæmorrhages. The eyes were hardened in Müller's liquid, and examined, for the first time after death, as no opportunity for ophthalmoscopic examination had occurred during life. There were no changes in the optic nerve nor in the choroid, save the compression of one retinal vessel in the disk by an accumulation of corpuscles, as noted by Leber. The retina was beset with dull points, ranging from the minutest size up to 2 mm. They increased in number towards the periphery. They were mostly of a round form, and prominent, so as to give a warty appearance to the retina, which appearance was also to be seen on the back of the coat when it was lifted from the choroid. These were evidently hæmorrhages, thrusting aside the layers, or lying between them; sometimes in the fibrous layer, sometimes between the granule layers, sometimes among the rods and cones, while few hæmorrhages occupied the whole thickness of the coat. Often two or more of these little lumps lay closely side by side, or above and below, being separated only by a minute but complete barrier of tissue. One fortunate section in the nerve layer showed the attachment of one of them to an underlying vessel. Another peculiarity also was generally observed, namely, that the red corpuscles were placed peripherally, while the middle consisted only of leucocytes.

This interesting record of Dr. Reineke, then, shows us nothing of irritation, but many of the so-called leukæmic tumours, masses which were, however, clearly referable to hæmorrhage rather than to lymphoid development, be this by adenoid or connective proliferation (Virchow), or be it by migration (Cohnheim).

Again, the close approximation without continuity of several of them, is suggestive of an accidental origin, rather than of a propagating process. The mutual relation in place of the two kinds of corpuscles is again quite identical with that seen in leukæmic hæmorrhages; and, indeed, for the matter of that, in many so-called 'softening' thrombi likewise, and it probably depends on the earlier death of the red corpuscles.

We are led, then, by sufficient evidence, to give up the fascinating hypothesis of the formation of adenoid tumours in

the retina of leukæmia, and to fall back on the belief that leukæmic, like albuminuric retinitis, is a combination of hæmorrhagic with irritative events, but that in both diseases we are as yet in want of further evidence as to the mode and causation of the latter.

This much seems to be a fair deduction from the cases already observed, speaking, that is, from the anatomical point of view alone, that in leukæmia there are three kinds of change in the retina which are in some measure distinct, and in some measure found together: the first and principal change is hæmorrhagic, the second is simply irritative and connected closely with disorder of the circulation, and the third consists perhaps in the peculiar and specific products of leukæmia. These latter have as yet been found in the retina by Leber only, and in the choroid by Engel-Reimers and Dr. Roth, for the lymphoid infiltration of the walls of the retinal vessels, if confined to the narrow limits in which it has occurred hitherto, can scarcely be referred to this special class of changes, but rather forms a part of the irritative changes, together with the hypertrophy of Müller's fibres and the fatty degeneration of the vessels which we frequently find together in other kinds of retinitis; such, for instance, as the albuminuric. To this latter class undoubtedly belong also the sclerosed nerve fibres, which are found likewise in the albuminuric and other irritative affections of the retina,—affections which present, in the midst of great variety, one uniform condition, namely, an excessive disorder of the circulation, whether systemic, intracranial, or orbital. The considerable dilatation of the marginal vacuoles seems to be a consequence of chronic hyperæmia of the retina. The jelly-like corpuscles of the choroid are likewise of irritative origin, and so often occur in senility, that in Dr. Roth's case they cannot be regarded as having any direct connection with this peculiar retinal disease. Finally, we must remember that while leukæmic and albuminuric retinitis agree in presenting hæmorrhagic and irritative characters, they agree also in this, that they both occur in conditions in which uric products are found in excess. In the former case urea and uric acid are formed in excess, in the latter they are insufficiently excreted.

CHAPTER IX.

ON THE RETINITIS ASSOCIATED WITH SYPHILIS.

IN my own practice, I seldom or never meet with syphilitic retinitis, numerous as are my cases of syphilitic neuro-retinitis¹. The retinitis belongs to an earlier period than the neuro-retinitis, and it is not associated, as is the latter, with intracranial proliferation (*vide* p. 106). I have, therefore, requested my friend Mr. Oglesby, who has great opportunities for observation, and who is a singularly patient and skilful observer, to write me the following notes upon the cases which he sees so commonly in Mr. Teale's practice and in his own.

Effusion of serum or lymph into the nervous textures of the retina is a characteristic feature of this disease. It not infrequently happens that during the earlier stages the ophthalmoscopic evidence is negative, from the fact that the effusion of serum is so slight as to escape detection even by the most practised observer; or, on the other hand, the deposition of lymph may be so great as to cause immediate and alarming symptoms: then the ophthalmoscopic evidence is at once apparent.

The pathological changes which take place are very interesting, more especially so when we are cognisant that such changes are also to be found in the retinae of those labouring under the inherited form of the disease.

Patients suffering from the early symptoms of this disease

¹ *Vide* cases reported by me in the fourth volume of 'St. George's Hospital Reports' for 1870.

complain of misty vision, objects appearing ill-defined in outline; bright light is shunned, and application to close work is followed by severe pain in the globe of the eye, temple, and brow.

Hyperæmia of the retina is one of the earliest symptoms of the disease; and as it occurs in an active form, its early recognition is of the utmost importance, so that means may be employed to stay, if possible, the inflammatory mischief which usually follows. The retinal vessels are numerous and enlarged, and the capillary tint of the disk heightened.

Effusion of serum into the nervous textures of the retina follows closely upon active congestion. It is characterized by the grey hazy look which it imparts, and is most readily seen when it encircles the disk, rendering its outline indistinct. The effusion poured out does not, as a rule, spread uniformly, but rather selects certain sections of the retina, and in no part is it to be found more frequently than in the neighbourhood of the yellow spot.

In those cases in which the effusion has invaded the entire membrane, we not uncommonly meet with turbidity of the vitreous humour, and often of a density sufficient to baffle any attempt at a thorough ophthalmoscopic examination. In such cases, the mapping out of the field of vision is of essential importance.

Should great infiltration of serum have taken place around the edges of the disk, tortuosity of the veins will be a prominent ophthalmoscopic appearance.

The effusion of lymph into the nervous textures is a symptom of serious significance. If it should take place with great rapidity, purulent infiltration of the entire membrane may result, with very rapid destruction of vision. On the other hand, if the deposition of lymph be gradual, and confined to isolated portions of the retina, the prognosis may be a hopeful one. In the latter condition, the patches, singularly enough, are frequently found in the course of one or other of the large retinal vessels. They somewhat resemble in appearance the bright spots seen in nephritic retinitis, and have not infrequently been mistaken for them. On close examination, there

is a marked difference, for they are not so brilliant in colour, and there is an evident peculiarity about their size and shape, which to a practised eye is easily recognizable. In a case of great interest, there occurred two patches of lymph, one slightly above the disk, the other immediately below, which had been mistaken for congenital nerve patches by more than one observer. This case occurred in a young man suffering from hereditary syphilis. Numerous cases of this kind are to be found from time to time in the out-patient room of an ophthalmic hospital ; and since the writer's attention was first drawn to the subject, he has been able to trace the disease through many of its phases.

Syphilitic retinitis is often present during acute attacks of iritis, and not infrequently follows inflammation of the choroid, or occurs simultaneously with that disease. Unlike the retinitis of albuminuria, it is often confined to one eye.

If the field of vision be carefully mapped in a number of cases, it will be found that peripheral contraction is a well-marked symptom during the early stages of the disease.

Hypertrophy of the connective tissue, deposition of pigment, and atrophy of retina, may all occur as sequences of the disease.

With regard to treatment, mercury is the most suitable remedy, and has a more rapid and certain influence in checking the disease than any other drug. Mercurial inunction is perhaps the least unpleasant manner of producing its specific effect. Next in merit stands the iodide of potassium, which, when given in large and increasing doses, often acts beneficially.

CHAPTER X.

ON THE AMAUROSIS OF DIABETES.

THAT cataract commonly appears in the course of glycosuria is well known; it would seem to depend upon the physical reaction between the sugar in the blood and the tissue of the lens, so that the two phenomena, glycosuria and cataract, rise and fall together. (Seegen.) The cataract is not likely to be overlooked by patients or by the medical adviser, and it needs little further consideration in this place.

It is not so well known that lesions of the fundus are also found in glycosuria, but changes, both atrophic and inflammatory, are described by several observers, such as Gräfe¹, Lecorché, Galezowski, Bouchut, and others. Some of these writers describe inflammatory changes in the fundus which bear a suspicious resemblance to albuminuric retinitis. It is likely that some error of diagnosis has crept in here, and that the observers, whose attention was given chiefly to the eye, had not sufficiently investigated the general symptoms. Actual renal disease is not infrequently associated with glycosuria, and albumen might, perhaps, have been found in the cases to which I refer, had not the presence of the sugar prematurely satisfied the analyst.

In future, any appearance of retinitis ought to sharpen the search for albumen.

Atrophy of the optic disks, on the other hand, does undoubtedly occur in a sufficient number of cases of glycosuria to make the co-existence seem more than accidental. Gräfe

¹ 'Deutsche Klinik,' 1859, No. 10.

(*loc. cit.*) was, I believe, the first to note the connection, and many cases have since been published.

I have found it in one case out of five which I have examined. I see that Professor Seegen, of Leipzig, in his new work², includes diminution of visual power among the symptoms of diabetes mellitus; and if the connection be regarded as established, it may be of great importance. It admits of being viewed in two lights: first, in the light in which Professor Seegen views it, namely, as a mere expression of exhaustion from innutrition; secondly, in the more interesting light which I am tempted to throw upon it, namely, that the nerve atrophy is but one part of that mischief in the central nervous system which probably lies behind the disorder of the liver as a cause of glycosuria. Recent observation, both experimental and clinical, points to the belief that glycosuria is, as a disease, a disease of the nervous system. It is clear that the fatty and other changes found in the liver after death are inessential; and such causes as mental strain or distress which seem to produce many cases of diabetes, would act rather upon the nervous centres. Heredity, again, which is known or believed to have an important place in the establishment of glycosuria, points rather to the nervous system, for heredity is so commonly observed in diseases of the nervous system; moreover, heredity in glycosuria is by no means always seen as such, but antecedent forms of disease, more obviously nervous, are often seen in the parents or relations. The last diabetic patient who came under my care was one of four brothers; the three who survive him are all of peculiar nervous temperament, and one of them is actually of unsound mind. All three are still young, and will probably suffer more seriously from nervous disease.

Still more important evidence is obtained from the marked tendency to obvious nervous disease in diabetics themselves. I need not enumerate the many cases on record of the co-existence of disease in the nervous centres with glycosuria. Among references immediately at hand is a case of cysticercus

² 'Der Diabetes Mellitus, auf Grundlage Zahlreicher Beobachtungen dargestellt, von Dr. Seegen, Prof. &c., Leipzig.' Weigel, 1870.

in the posterior brain accompanied by glycosuria, reported by Cyon; a like case, published by Rosenthal in his new volume (foot-note, *loc. cit.* p. 66), where diabetes mellitus and atrophy of the optic disk (observed by Jäger) were among the symptoms of a tumour at the base of the encephalon; observations on palsy of the soft palate in diabetes recorded by Dr. Sanders; and an essay, founded on several cases, by Dr. J. W. Ogle, in the first volume of the 'St. George's Hospital Reports.' Dr. Dickenson has also collected and, I believe, published several instances of the same kind. A patient of Dr. Bronner, of Bradford, came to me some months ago with a long history of glycosuria associated with chronic degenerative disease of the cerebro-spinal centres, the precise nature of which is uncertain, and, fortunately for the patient, is as yet out of reach.

In Dr. Rosenthal's case, it seems probable that the optic atrophy was a direct result of the basilar tumour, and in no intimate association with the glycosuria; but if we find, as I think I may say is the fact, that glycosuria is intimately connected with central nervous lesion, and that atrophy of the disks is often associated with glycosuria, it becomes a matter of deep interest to us to know whether the optic atrophy may not be regarded as an outlying part of the central mischief.

Unfortunately I have been disappointed in the autopsies which would have enabled me to give an opinion on this point; but when, after the vain endeavours of two or three years, I mentioned the matter to Lockhart Clarke, that wonderful observer immediately produced some slides displaying granular disintegrations about the calamus scriptorius, removed from a diabetic patient,—a discovery which harmonizes more exactly with experimental results than clinical and pathological observations are wont to do.

CHAPTER XI.

OXALURIA.

As cases are published of optic change co-existing with oxaluria, I think it well to draw attention to them. In the 'Ophthalmic Review' (vol. i. p. 213) a neuro-retinitis is described by Mr. Mackenzie as due to oxaluria. Turbidity of the vitreous is also described as having a like origin. The administration of the mineral acids is said to have brought about an improvement. Mackenzie believes that the neuritis is due to a poisoning of the blood by way of a faulty digestion. The matter has, indeed, two aspects: first, we may inquire whether the accidental presence of oxalates is able of itself to injure the retina by a physical reaction of the kind of osmosis, or by means of such irritation as oxalic acid produces in tissues with which it comes in contact; or we may ask, whether the neuritis and the oxalates are both evidences of some anterior and peculiar constitutional state?

The first question I leave open, as I have no evidence to lead me to a reply. As to the second, I would remind the reader that oxaluria is no longer regarded by physicians as significant of a particular group of changes, but is a symptom unattached, which may present itself among many various groups. Either, then, neuro-retinitis may be caused by the direct action of oxalic acid, however formed, upon these tissues, which is too important a supposition, both in itself and in its remoter bearings, to be slightly dealt with; or the oxaluria was but a subordinate phenomenon in some disease, perhaps of the nervous system, in which neuro-retinitis also appeared as an independent event.

CHAPTER XII.

TOXIC AMAUROSES.

SECTION I.

Amaurosis from Alcohol Poisoning.

It is easier to ascertain the effects of alcohol upon the nerve of sight than those of tobacco, because the other results of alcoholic poisoning are better known, and its pathological consequences more familiar to all. There is accordingly more agreement among ophthalmic surgeons concerning the effects of alcohol than concerning the effects of tobacco, the evidence of over-use in any given person being more obscure in the latter than in the former case. Observers agree in believing that prolonged alcoholic intoxication exercises a distinct and primary effect upon the optic nerve, in addition to its other consequences.

It seems almost certain that the nerve may not only suffer indirectly as a consequence of intracranial degenerations, but that it presents primary and independent changes of its own, which may or may not march *pari passu* with the like changes in the cerebro-spinal axis. A study of the effects of alcohol upon the optic nerves is therefore full of interest and instruction, being, as they probably are, but a visible part of those going on in other districts of nerve tissue which lie beyond our sight. In speaking of alcohol, I use that word alone, as it seems probable that the mischievous effects of drinks of this kind are, for the most part, in proportion only to the amount of alcohol they respectively contain. I may

mention, only to dismiss with a few words, a kind of amblyopia, not at all uncommon in drunkards, in which there are no obvious changes in the fundus. As in uræmia, there is an amblyopia potatorum with ophthalmoscopic signs, and an amblyopia without them. This latter defect is not rare, and may be due to a congestion of the choroid, with consequent pressure upon the rod layer of the retina. This can only be guessed at from the well-known tendency of alcohol to cause congestion of the blood-vessels of the head and face, for I do not think that any observer would venture to say that he could distinguish moderate and simple hyperæmia of the choroid with the mirror. It does, however, seem certain, from much testimony, that this amblyopia is quickly relieved by local bleeding.

The other kind of amblyopia seems to depend upon the same congestion, with consequent tissue deterioration in the optic nerves, which we see also in the brain of drunkards, and which causes the opacity of the pia mater and arachnoid of such persons. The action upon the highly vascular optic nerve seems to me to be parallel to this action upon the membranes. My own experience bears me out fully in saying that congestion of the vessels of the disk and retina is very common in patients who present themselves with symptoms of alcohol poisoning, which class of persons appear in large numbers in the out-patient consulting-rooms at the Infirmary. I always examine the fundus in cases of delirium tremens, as soon as restored tranquillity will allow me to do so, and in the great majority of cases I find congestion and opalescence of the disk and full retinal veins. Sight often remains unaffected, or its dimness passes unheeded as a part of the headache and mental confusion¹. When I worked in Mr. Teale's eye-clinic, I met with many of this kind of persons whose

¹ Galezowski has published a number of observations of colour blindnesses as seen in disease of the nervous parts of vision. I have not discussed them anywhere, as I have not made any adequate examination into the matter, and Leber has shown in the fifteenth volume of the 'A. f. O.' that such functional disorders are of no use in warning us of commencing or of threatened atrophy. When therefore the sight-fields are complete, as in alcohol poisoning, the colour tests are of no diagnostic value.

congestion had passed on into low proliferative action with atrophic contraction, the disk becoming reddish grey, then greyish blue, and finally greyish white. The fine vessels also vanish, but the larger veins of the retina remain longer in a state of fulness.

I much doubt the value of the recent experiments which are said to show that alcohol, opium, and chloroform attack the myeline, coagulating it, as it were, and breaking it up i..to minute brilliant points.

Changes like these so soon arise in the course of the preparation or decay of the parts examined, that such assertions can scarcely expect as yet to receive much credence². I believe that the alcohol acts rather as an irritant to the highly vascular brain and optic nerves, as it does to the liver, and, *pace* Dickenson, the kidneys and arteries, setting up proliferation of the connective elements with contraction and atrophy. Among the symptoms which lead one to examine the fundus in drunkards, are loss of strength and appetite, coated or raw tongue, vomiting, confusion of mind, loss of memory, trembling of the lips, and disturbed sleep.

SECTION II.

Tobacco Amaurosis.

It is very difficult to discuss the well-known proposition, that the excessive number of cases of amaurosis occurring in the male sex are due, in great part, to the smoking of tobacco by that sex. While, on the one hand, observers who form this opinion are bound to state it; yet, on the other hand, it is so difficult to isolate the antecedents of amaurosis in men, in order to distinguish those which are essential; and, again, if the efficiency of several antecedents be estimated,

² Nor, on the other hand, can I attach much importance to the negative results of Dr. Paul Ruge, of Berlin, who examined the effects of alcohol upon many animals. The important condition of the long-continued use of alcohol is not present in such experiments. So far they differ from the pathological results of myself and many others.

it is so difficult to say which of several true causes is the actual cause, that those who, like myself, have not the opportunity of pursuing the facts themselves on a large scale, are reduced to the somewhat invidious task of comparing the qualifications of those who have such opportunities, and who pronounce accordingly on one side or the other. After what I have said concerning the action of alcohol, for instance, the reader will see how hard it is to separate the two agencies, alcohol and tobacco, in their effects upon the nervous system, the use of the two being so commonly found in one patient.

One thing is clear, and must have a certain weight, namely, that a very considerable number of our best observers, both at home and abroad, and observers who have had large experience, express themselves very strongly, if not very decidedly, upon the efficacy of the use of tobacco to cause amaurosis.

Before the ophthalmoscope was discovered, Mackenzie spoke as follows: 'I have already had occasion repeatedly to hint my suspicion that one of the narcotico-acrids which custom has foolishly introduced into common use, namely, tobacco, is a frequent cause of amaurosis. A majority of the amaurotic patients by whom I have been consulted have been in the habit of chewing, and still oftener of smoking, tobacco in large quantities. It is difficult, of course, to prove that blindness is owing to any one particular cause, when, perhaps, several causes favourable to its production have for a length of time been acting on the individual; and it is especially difficult to trace the operation of a poison daily applied to the body, for years, in such quantities as to produce at a time only a small amount of deleterious influence, the accumulative effect being at last merely the insensibility of a certain set of nervous organs. At the same time, we are familiar with the consequences of minute portions of other poisons which are permitted to operate for a length of time on the constitution, such as alcohol, opium, lead, arsenic, mercury, &c.; and we can scarcely doubt that a poison so deleterious as tobacco must also produce its own peculiar injurious effect³.' Well and carefully expressed as is this passage, we cannot but see

³ *Loc. cit.* p. 888.

that Mackenzie had a strong tendency on *a priori* grounds to anticipate the results which he describes; and we are disposed to inquire how large a percentage of patients suffering from whatever other kind of disease are smokers? When, again, a person is described as a great smoker, unless we have some definite information concerning the quantity of tobacco used, we are obliged to remember that the standard of moderate smoking varies according to the prejudices of the physician. A patient was sent to me from a distance the other day as a sufferer from the effects of excessive smoking; but taking one week with another, I could not make out that more than one ounce of the weed was used in that time.

There is, moreover, this important and curious fact to be remembered, that many substances which are intensely poisonous in concentrated forms, are nevertheless innocent, if not useful, when administered in repeated and feeble doses. The experiments upon lower animals with the essential oil of tobacco, which are quoted by Mackenzie in support of his opinion, are by no means strong evidence of the likelihood that any similar effect would be produced by ordinary smoking. To use the words of C. S. C.,

‘Cats may have had their goose
Cooked by tobacco-juice;
Still, why deny its use,
Thoughtfully taken?’

I am not an habitual smoker myself, but I can scarcely avoid the conviction that it is decidedly useful to many of my acquaintances, and seems to be harmless even to those abandoned Turks and Teutons ‘who smoke perennial pipes and spit.’ The increase of amaurosis, indeed, seems to be like the increase of insanity—it appears in numbers corresponding to the capacity of the institutions prepared for its reception, so we cannot be sure that its frequency in our ophthalmic clinics is any consequence of the wider use of tobacco. Where we have, if possible, to isolate any one condition, such as tobacco smoking, we can only hope to eliminate accidents by including very large numbers, not of amaurotics, but of

smokers, in our survey, and comparing them with like numbers of abstainers.

Mr. Carter is the only observer, so far as I know, who has seen the necessity of handling the question widely in this way. Mr. Carter, in his well-known work founded upon that of Zander, compares the experience of the English with certain Eastern physicians. 'I was myself,' he says, 'for many months resident in various parts of Asiatic Turkey, and had opportunities of becoming acquainted with the prevalent diseases of the country, among which, so far as I saw, amaurosis could not be numbered.' Mr. Carter also received the following additional information. Mr. Farquhar, for many years surgeon to the British Consulate at Alexandria, says: 'In answer to your inquiry respecting amaurosis, I can only say, that during the whole of my residence in Egypt, and among the many thousand diseased eyes which I examined, it was always a mystery to me that I saw so few cases of this affection. The Egyptians, if it be possible, smoke even more than the Turks.'

Dr. Dickson, the physician to the British Embassy at Constantinople, writes: 'Amaurosis, taking the term in its widest sense, is not a common complaint in Constantinople, or in Turkey generally; and yet smoking tobacco is so prevalent a vice, that it is practised by the whole population, Mohammedans, Christians, and Jews, with hardly a single exception. The usual amount consumed by one person, per month, may be estimated at $2\frac{1}{2}$ lbs. avoirdupois. In addition to my own testimony, I may add that of Dr. Millinger, one of the oldest and most celebrated physicians here, who declared to me that amaurosis was a rare affection in Constantinople.' Dr. Dickson adds, curiously enough, 'During a ten years' practice at Tripoli, I found that amaurosis was a common affection there, and yet the natives never smoke.'

Dr. Hübsch, the chief oculist in Constantinople, writes to the same effect, and in the course of a very interesting letter says: 'Je n'ai jamais pu attribuer l'amaurose à l'abus du tabac; le nombre des fumeurs est immense, le nombre des amauroses est limité.'

If we turn, again, to the workers in tobacco manufactories, we are unable to find that amaurosis occurs with any undue frequency among them, although we do find distinct evidence of certain degrees of poisoning, as shown in disorders of the nervous system. In Leeds, tobacco is largely manufactured, but my own inquiries have failed to discover any prevalence of amaurosis, even among persons who have worked fifteen or twenty years at the occupation. Mr. Oglesby, of Leeds, a most industrious worker, with large opportunities of observation, tells me that in all his experience he has met with but two cases which seem to bear out the belief that amaurosis may be due to tobacco. These two cases are, however, so striking, that I have obtained his permission to publish them in the Appendix (Nos. 122, 123). The two cases seem to suggest that enormous use of tobacco may, in rare cases, so deteriorate the nervous centres, that the optic nerves suffer with other parts; but they do not, I think, help to prove that tobacco has any special and isolated effect upon them in cases where the rest of the nervous system betrays no symptoms of injury.

At the same time, it will not do to forget that the supporters of the belief in tobacco as a cause of amaurosis are among the greatest names in ophthalmic science. I have already cited Mackenzie, and next to him I may place Sichel, who speaks strongly to the same effect⁴.

Among recent English writers, Mr. Wordsworth and Mr. Hutchinson have urged the same opinion, and Mr. Critchett is understood to have expressed himself in like manner.

It is worthy of note, that the German observers speak very cautiously and doubtingly concerning this effect of tobacco; and although our own writers have quite as much claim to be heard as those of Germany, yet, on the other hand, it must be remembered that the German oculists practise in the midst of people incessantly occupied in smoking pipes of very strong tobaccos.

Nor has Sichel found many decided supporters in France. Follin, in his article 'Amaurosis,' in the *Dictionnaire Ency-*

⁴ 'Influence du tabac sur la vue,' in '*Annales d'Ocul.*' 1865. And previously in '*Union Médicale*,' 1860-63.

clopédique, speaks with much hesitation as to the effects of tobacco; and of the two cases of the kind which he records, one died of well-marked cerebral disease, which, whether itself due to tobacco or not, was probably the direct cause of the amaurosis; the other is said to have recovered on laying his habit aside: but as people do not recover in this simple way from white atrophy, it seems more likely that the amaurosis was but an instance of that anæmia (*vide* page 50), which is a common consequence of exhausting causes of almost any kind.

While, then, it would be neither courteous, nor in the interest of our common purpose, to treat lightly such testimony as that of Mr. Wordsworth and those who agree with him, yet I may be permitted to indicate these three great difficulties which are inherent in the subject. First, as I have said, amaurosis is very common, and is common in certain districts especially, but in districts which do not coincide with the districts of the principal manufacture or consumption of tobacco. Secondly, that many causes are at work, in this present busy age especially, which may have far more to do with the causation of amaurosis: I may refer to the use of alcohol as one of these; as another, to the incessant over-use of the eyes in a large number of the competitive trades in which men especially are engaged; and again, to those many strains upon the mind and nerves which overtask the nervous centres, and carry the optic nerves with them. Thirdly, that in many cases of so-called recovery from amaurosis on omission of smoking, we have no certain evidence that the condition was not one of general anæmia, made especially manifest in that kind of loss of vision which accompanies menorrhagia, lactation, and the like. Unfortunately, mapping of the field does not help us much here, as in amaurosis from toxic causes the diminution of vision is often uniform all over the field. Fourthly, we have no satisfactory evidence that the cases quoted were well watched for any length of time. Degenerative nervous disease, such as locomotor ataxy, general paralysis, and disseminate sclerosis, are commoner in men than in women; and of such diseases, white atrophy of the optic nerve may be the

first sign : see, for instance, the case of MacCarthy, No. 79 in the Appendix. In ophthalmic clinics, the attention of the surgeon is given chiefly to the eye ; he has neither the time nor the habitual tendency to inquire into and make himself familiar with obscure forms of central nervous disease ; and any physician among my readers, who has been accustomed to turn over ophthalmic treatises and reports, will bear me out when I say, that in cases of optic neuritis, albuminuric retinitis, and the like, the poverty and inadequacy of the report of the medical details is too often so great as to make the case useless as evidence of anything. To no ophthalmic surgeons do these strictures apply with less force than to Mr. Hutchinson and Mr. Wordsworth ; but I cannot refrain from saying, that the description given by Mr. Wordsworth of the onset and course of the amaurosis of tobacco, and which he thinks is peculiar to that amaurosis⁵, is nevertheless the exact description of amauroses which have been watched throughout by myself in many forms of cerebro-spinal disease, and in general paralysis in particular.

SECTION III.

*Lead Poisoning*⁶.

The action of lead in the organism tends, as is well known, to produce a deterioration of the nervous system, and, moreover, to select certain parts of that system in a way which seems to us as yet to be capricious. The eye suffers as one of these parts, and it suffers in three ways. Firstly, the effects of lead may be seen in the ciliary muscle, causing palsy of accommodation : secondly, in the optic nerve, causing atrophy thereof, which atrophy is sometimes preceded by a stage of subacute irritation : thirdly, and indirectly, by causing granular degeneration of the kidney, with its peculiar retinitis.

The first of these effects is only indicated, as it lies beyond

⁵ Carter's 'Zander,' p. 132.

⁶ *Vide* plate prefixed to this chapter. These plates serve well, also, for illustration of some sections of the fourth chapter.

the subject-matter of this work ; and the third, which is, perhaps, the most frequent, has already been described in Chapter VII. We have only to deal, therefore, with the second, with atrophy of the optic nerve, preceded or not preceded by chronic or subacute neuritis. If complicated with palsy of the ciliary muscle, the case will be readily simplified by the use of an appropriate convex lens.

I have seen but one case of saturnine atrophy, and this was some time ago : I publish brief notes of the case in the Appendix, No. 120. Lead poisoning is very uncommon here, as there are no trades in Leeds which favour it ; the house-painters no longer grind their own colours in winter as they used to do, so that among them lead poisoning has almost ceased to exist. Many cases are recorded, however, in the journals, both home and foreign ; still, I am disposed to think that the event is one of the less common results of lead poisoning. Christison, for instance, makes no allusion to amaurosis as a result of lead poisoning. E. Meyer published in the 'Union Médicale' two cases, which were read at the Medical Society of the Elysée on Feb. 3, 1868 : in one of his cases there was white tendinous atrophy, with complete blindness ; but in the second, there was well-marked neuro-retinitis. This case is published in the Appendix, No. 117. In my own case, that of an out-patient of the Leeds Infirmary, there were grave symptoms of intracranial mischief-symptoms, which pointed to softening, complicated with some hæmorrhage. In Meyer's case, also, there were serious cerebral symptoms. In these instances, it may sometimes be difficult to say whether the optic atrophy is direct or indirect ; whether, that is, it succumbs, like the encephalic parts, to the direct action of the lead, or whether its changes be merely symptomatic of intracranial lesion of whatever causation.

Saturnine amaurosis was noticed in præ-ophthalmoscopic ages ; the earliest reference which I have in my possession being to Duplay, in the 'Archives Générales de Médecine, ii^e série, tom. v. p. 5. Paris, 1834. I have not seen the original ; the reference is copied from Mackenzie.

It is to Mr. Hutchinson, however, that we owe the most

important essay on saturnine amaurosis. This essay was published, with illustrations, by Mr. Hutchinson in the seventh volume of the 'Royal Ophthalmic Hospital Reports.' The number appeared while these sheets were in the printer's hands, and I am enabled by the kindness of the author to reproduce his illustrations in this place. Mr. Hutchinson gives notes of five well-marked cases, four of which were under his own care, and the fifth under Dr. Charlton, of Newcastle. The first case, in a young woman, aged 19, had been described in the preceding volume of the 'Reports,' and is thence quoted by me in the Appendix, No. 119. The second and following cases are so important, both in themselves and in their bearings upon the whole question of lead poisoning, that I venture to quote them in their entirety.

CASE II.—*Double optic neuritis in connection with lead poisoning, but without other symptoms—Constitutional symptoms of neuritis ill-marked—Description of the disks.*

William Argent, æt. 40, has been a house-painter for twenty years past. He is now a pale complexioned man, and around his teeth is a very marked blue line. He asserts, however, that he has never suffered from lead colic, nor from any other illness which he attributed to the paint. In November, 1867, he was laid up for three weeks with 'rheumatics,' and had swelling of the backs of hands, great toes, and ancles. Often before that he had suffered from rheumatic pains in various parts. His sight has never been quite what it was since this illness. Two or three months ago, however, it failed more decidedly, and he now found that his left eye was much worse than the right. He has for the last few years often suffered from pain in the head, which he considered rheumatic; it appears to have been a frontal neuralgia. At the time his sight was decidedly failing he had not more headache than usual, nor was there much tendency to sickness. During the last two or three months he has, however, almost always had sickness (to vomiting) once or twice a week. This is a symptom quite new to him.

He applied to me first on February 8, 1869. I found that with his left eye he could only see to spell out No. 16, and with the right No. 4. The disk of the left was very pale, and of a blue-white; whilst the vessels were much reduced in size, the arteries being so small that they could scarcely be traced. The margins of this disk were quite clear. The disk of the right was white in its nasal (inverted) half, and on the outer part was covered with pale lymph, which concealed its edges. The quantity of lymph, however, was very much less than is usual in many other forms of neuritis. Although headache and sickness have been but slightly marked during the neuritis, yet it is quite certain that they have been present to some extent.

In the left eye the margins of the disk are very definite, not in the least jagged. There is a single minute dot of pigment on the inner side, but with this exception, there is no evidence of disturbance of the choroid. The retina is quite clear at all parts. In the right there is no evidence of choroidal implication. The retina is hazy to a slight extent round the disk, but not to any great distance.

His smell and his tactile sensibility seem perfect.

He has sometimes seen flashes and sparks of light before his eyes.

Reference to Plate.

Figs. 1 and 2 in the plate show the ophthalmoscopic appearances in the two disks of this case.

Amongst the peculiar ophthalmoscopic features of plumbic neuritis we may mention (*a*) the small amount of lymph usually present; (*b*) the absence of colour in the lymph; (*c*) the absence (not invariable) of extravasations of blood; and (*d*) the early and great diminution in size of the arteria and vena centralis. The choroid does not appear to be in the least implicated.

CASE III.—*Acute lead poisoning with optic neuritis—Recovery of health, but with permanent blindness.*

Mrs. Driscoll, æt. 25, came to us at the Moorfields Hos-

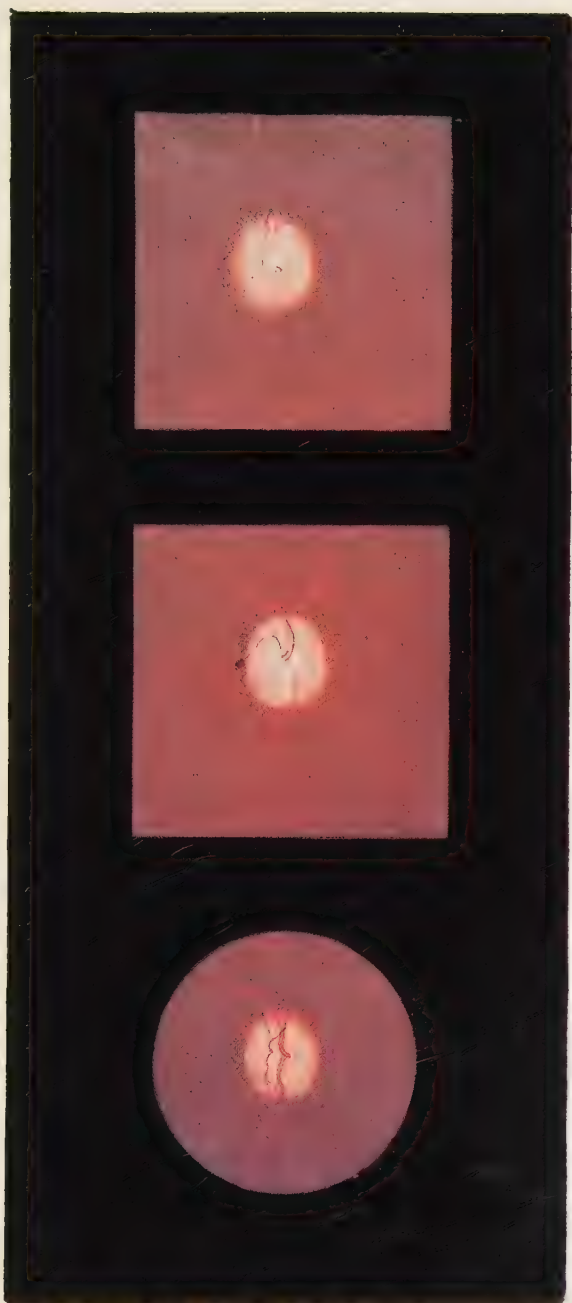


Fig. 1.

Fig. 2.

Fig. 3.

E. Burgess del adnat d. ch. lith

W. West imp.

NEURITIS FROM LEAD-POISONING.

pital, January 7, 1870, having been almost totally blind since an acute attack of lead neuritis four years previously. The evidence she gave us was the following. She went to work four years ago in a lead factory, being at the time in perfect health, and having never before been employed in that way. After six months' employment in the factory she became very ill, had pain in the head, and severe sickness. Her illness was attributed to her work in lead. She was confined at home for a month with these symptoms, and became at the same time almost blind. The sight failed suddenly and quickly. She had no dropped wrist. When she recovered from her illness the state of her sight was such that she could not return to work. Since that time (four years) she has never been exposed to the influence of lead, and has enjoyed fair health. Her sight has, however, scarcely improved at all.

*State of vision and ophthalmoscopic appearances four years after the attack (January 4th, 1870).—*She cannot distinguish a gas-flame in a dark room, but she asserts that sometimes she can just see light. Both eyes are alike. She is of dark complexion, but pale. With the ophthalmoscope the conditions in the two eyes are found to be exactly alike. The media are quite clear, and the fundus is easily seen. In each the disk is quite white. The arteria centralis is diminished to perhaps half its natural size, the veins being much larger. The lesser vessels, which give pink tint to the normal disk, have wholly disappeared. Near to the disks the trunks of the vessels are decidedly obscured, as if from past inflammation, and there are in places white lines along their trunks. (Fig. 3 in plate.)

CASE IV.—*Hereditary gout increased by lead poisoning—Neuritis of the left optic nerve in 1867 resulting in permanent blindness—Neuritis of right optic nerve in 1870 (still under treatment)—Interesting facts as to asymmetry of symptoms.*

John West, æt. 44, a painter by trade, was admitted under Mr. Hutchinson's care at the Royal London Ophthalmic Hospital on September 29, 1870. About three years before

this he had been under Mr. Dixon's care for failure of his left eye, when he was told that he had 'atrophy of the nerve.' This eye became at that time all but blind. He applied on the second occasion on account of the right eye, which had been failing gradually for about three months.

On examination, his vision was found to be, for the *right* eye, 20 J. (increased by a convex glass to 7 J.) and $\frac{20}{200}$; with the left he could only see the light. There was contraction of the outer part of the field in the right eye. With the ophthalmoscope it was found that the *right* disk was of a dirty grey colour, its margins 'fluffy' and ill-defined, and the large vessels somewhat diminished. The left disk was in a condition of advanced white atrophy. A blue line was found on his gums, and the gums were much wasted.

The interest of this case lay in the *cause of the neuritis*, and in the occurrence in *the same patient of optic neuritis, lead colic, and gout*. His family and personal history was as follows:—His paternal grandfather was 'a martyr to gout;' he was a stout man, a hairdresser, and drank beer. The patient's father was a plumber, and he also suffered from gout. The patient, a painter, had his first attack of gout sixteen years before admission, in his left great toe; since that time he had had many attacks; the left side seemed to have suffered more severely than the right, and he stated that his left arm and leg had often been rendered quite useless by the gout for a month or two together. His attacks of gout had usually been unsymmetrical; they came on suddenly, and often at night; the affected joints were swollen, shining, red, and painful. He had suffered repeatedly from colic, but never from 'wrist-drop.' As to the cause of his attacks of gout, he said that he had never drunk much beer, and but very little spirits; he had always found that an attack of gout came on after he had been 'flatting' colour.' In 'flatting,' the work is done in closed rooms, the colour is entirely lead, and is mixed with pure oil of turpentine,—a liquid which evaporates quickly, and probably in this way carries off more vapour of lead in a given time than the less volatile liquids used in ordinary painting. He stated that the 'flatting' often produces a sort

of intoxication, especially in the men who work highest up in the room, and is frequently followed by attacks of colic. He himself had had no colic for five years, and he attributed this exemption to his having worn a moustache during that time.

It is interesting to notice, that the eye which failed first was on the side of the body which was earliest and most severely attacked by gout.

One of my most instructive cases in reference to this subject came under my notice at the Newcastle Infirmary, during the visit of the British Medical Association last August. The patient was under Dr. Charlton's care, and I am indebted to him, not only for permission to examine it, but also to make public use of its facts. Dr. Charlton told me that he had seen several cases of loss of sight from the effects of lead.

CASE V. — *Acute lead poisoning, with imbecility and general paralysis—Double optic neuritis—Complete and permanent blindness—Recovery of other functions under treatment by iodide of potassium.*

Kate Morgan, æt. 19, came to Newcastle in September, 1869, and engaged herself in the lead works. Her occupation consisted in carrying white lead. In about four months she began to suffer from symptoms of poisoning. Her bowels became costive, and her muscles weak. Her sight also began to fail, and in five weeks from its first defect she was quite blind. When, in the beginning of May, she was admitted into the infirmary under Dr. Charlton's care, her state was most deplorable. She was wholly blind, unable to speak distinctly, paralysed in all limbs to the extent that she could not use them, and liable to incontinence of both urine and fæces. Her expression was that of an imbecile, and, in addition to her defect in vocalisation, there seemed also to be inability to find her words. There was a distinct blue line around the gums.

When I saw her in August, she had been three months

under treatment by iodide of potassium and sulphate of magnesia—the former in full doses. She had for six weeks been able to leave her bed, and she could now walk well. Her face had regained an intelligent expression, and she could speak clearly. The incontinence had disappeared. She remained, however, still quite blind. Dr. Page, the house-surgeon, who supplied me with the above facts as to her case, kindly procured me also an opportunity for ophthalmoscopic examination. I found the disks both in the same condition of atrophy after neuritis. They were blue-white, of a somewhat dirty appearance, and the central vessels had much diminished in size. There were still traces of lymph about the main trunks. At a distance from the disks there were no deviations from the healthy state.

In this case it is evident that the poisoning went very near to a fatal termination.

The girl had probably been very careless in her habits, and had thus obtained an unusual dose of the metal. The result of the treatment was most marked, and most satisfactory. If we ask why the other nerves recovered their functions so much more fully than did those of sight, we shall be obliged to give a hesitating answer. Possibly the strength of the sheath surrounding the optic nerves renders inflammatory swelling more injurious to them than it is to others. It is possible, however, that the cause of the paralysis of the limbs was a central inflammation rather than a neuritis. As to what had occurred in the eyes, there could be no room for doubt, especially when we compare it with the other cases which I have recorded.

SECTION IV.

Hypnosis and Narcosis.

The reader has seen, from much that I have already said, that we cannot, as yet at any rate, be satisfied to take the disk as a dial-plate which exactly and visibly indicates the states of the encephalic circulation. At the same time, if we

can properly estimate the accidents which modify the circulation of the disks, such, for example, as the correlative degrees of intra-ocular tension, we may be able to get some knowledge from it regarding the variations of blood-pressure in the encephalon during the operations of certain general disorders, of sleep, of narcotic poisons, and so forth.

Evidence of this kind, if really trustworthy, would be invaluable, both in clinical work and in physiology. At present, after studying many cases of general anæmia, and of presumed cerebral congestion, such as in alcoholism, I have been forced to believe that anæmia and congestion must be not only considerable, but also much prolonged, if it is to map itself out in the fundus of the eye. At the same time, the changes which have been observed in the eye during sleep, by Dr. Hughlings Jackson, are of a more positive character. The observations seem, indeed, to be conclusive, as far as they go; and I shall, therefore, now quote them in Dr. Jackson's own words:—

‘It is scarcely necessary to say, that my reason for examining the eye during sleep was to help to form some idea as to the condition of the circulation in the brain itself in this physiological condition; the retina and the brain being supplied by branches of the same trunk, the carotid, and these by the same vaso-motor nerves. We may consider the retina as part of the brain extruded through an opening in the skull.

‘Still, I have drawn no conclusions as to the condition of the circulation of the brain in sleep, as the subject requires to be studied on a very extensive basis, of which these observations can form but one part. Indeed, I study the physiology of sleep, in order to learn somewhat as to the circulation in the brain in certain allied pathological conditions. For instance, I am anxious to know the condition of the veins and arteries of the brain in the profound sleep, or, perhaps more correctly, in the stupor which follows a paroxysm of epilepsy. I have examined the retina in this condition, and several times in cases of severe congestive headache after a fit. The results of these and of many other

examinations in cerebral cases, I hope to have the pleasure of giving in some future number.

‘A girl, aged 11, was admitted into the Hospital for the Epileptic and Paralysed, under the care of my colleague, Dr. Brown-Séquard, for hemiplegia, which had existed several years. Of this, at the time when the following observations were made, there was little or nothing left, and the child was in fair general health.’

I give the following extracts from my diary as the simplest way of recording several observations :—

‘Sept. 3rd.—I tried, first, to examine the eye without using atropine; but the pupil was so small, as is usual in sleep, that I could not illuminate the fundus. I therefore dilated *one* pupil by atropine, and then examined the fundus of both eyes when the child was awake. I found the optic disks normal. They were equally well coloured, but not abnormally so. I had examined her sight carefully before dropping in the atropine, and found it perfect. When in deep sleep, one pupil was contracted; that dilated by atropine remained enlarged. By the aid of a very intelligent nurse, who held up the upper lid, I was enabled to examine the optic entrance, to which, for the present, I confine my observations. I found that the optic disk was whiter, the arteries a little smaller, and the veins larger than in waking. The veins were thick, and almost plum-coloured. The neighbouring part of the retina, also, was more anæmic.

‘Sept. 6th.—The pupil was now rather small. I saw the optic disk steadily, and could confirm my first statement. The arteries were certainly smaller, and the veins larger than in waking. The other parts of the optic disk were whiter, as was also the neighbouring part of the fundus. She had been well tired by a long romp with the nurse.

‘Oct. 3rd.—The pupils had regained their normal size. I again put atropine in the right eye, and examined with the ophthalmoscope. I carefully noted each vessel, especially the smaller ones, and learnt by heart the position and size of both veins and arteries, and also the condition of the optic disk as to colour. At night I examined the eye during sleep.

The pupil was smaller than when the child was awake, but I luckily saw well for a long time. The optic disk was not so red, the arteries were certainly smaller, and on this occasion, I think, the veins were no larger, and about the same as when the child was awake. I then roused her, and examined under similar conditions of light, position, &c. She was awake, but sleepy. I found that the arteries were larger; but, on looking again, I found them smaller, as in sleep. They alternated several times. I could not long dwell on the disk; and my opinion is, that the alternation was gradual.

‘Oct. 16th.—A girl, aged 11, a patient under the care of my colleague, Dr. Ramskill. I dilated the right pupil with atropine. In sleep, it remained dilated; the other was contracted. I saw well in this case. The child was deeply asleep, and I had the optic disk under view for a long time. All I can say is, that the disk itself was rather paler in sleep. I roused the child till she was fairly awake, and the only difference then was, that the disk was a little redder.

‘The pupil, under the influence of atropine, dilated to the fullest extent when awakened; twice the size it was when the child was asleep. The contraction was not due to the light only; it was the contraction of sleep.

‘Oct. 21st.—I put atropine in the right eye, and dilated the pupil to the fullest extent. The child is a somnambulist, and I found her in the ward at 10 P.M. in the arms of the nurse. The left pupil (the one without atropine) was not so small as usual in sleep. The other was as large as it was when she was awake. She was apparently asleep, however. I examined the eye, and then fairly awakened her, by pinching and making her speak and getting her to look in certain directions. I again examined her eyes, when she had gone to sleep in bed. I feel convinced that the arteries were a little smaller, and the veins larger. I saw well, and for some time.

‘Oct. 24th.—Atropine as usual. I saw well; the disk was whiter, and the arteries smaller.

‘I ought to observe, that in all these examinations the dif-

ference in the size of the arteries and in the coloration and the optic disk during sleeping and waking was but slight.'

I have been unable to repeat these observations. In the few cases where an opportunity has offered itself, the uplifting of the eyelids and the glare of the reflected light always awoke the person to be watched. The way in which Dr. Jackson speaks, however, and his well-known caution as an observer, command the greatest weight for his statements; moreover, we must remember how closely these results accord with those obtained by Hammond and Durham. If cerebral function depend upon an adequacy of arterial blood, this function cannot be carried on under a deficiency of arterial blood, whether such deficiency be due to emptiness of the vessels, or to their repletion with blood unærated.

So no doubt we have sleep, artificial or natural, due to anæmia; and sleep, artificial or natural, due to venous congestion of the capillaries.

It may be not only very interesting to us as physiologists, but very important to us as therapeutists, to classify our agents according to the two ways in which they act upon the brain; and to classify in like manner the various states of sopor not induced by drugs, in which our patients may be found. May not coma be thus contrasted with true sleep, and bromide of potassium with alcohol? I am led to these reflections by the fact discovered by Dr. Lewizky, of Kasan, and published in Virchow's '*Archives*,'⁷ that bromide of potassium causes retinal anæmia.

As Dr. Lewizky's observations seem to have escaped the notice of ophthalmologists, and as I have verified them in two patients who were under the influence of large doses of bromide of potassium, I extract the following lines from his article.

Dr. Lewizky says, after describing certain observations upon the vessels elsewhere, and showing that bromide of potassium causes a narrowing of the vessels: 'On a rabbit in which I trepanned the top of the skull, and observed the

⁷ Vol. 54, Pt. ii. p. 193, part of article '*Ueber die Wirkung des Bromkalium auf das Nerven-System.*'

vessels of the pia mater, it seemed to me as if the membrane became paler and some of the fine vascular branches diminished. In some ophthalmoscopic observations which I made upon the vessels of the retina with regard to the influence of bromide of potassium it was quite evident (*ganz klar*) that these vessels grew narrower⁸. This is very coherent with Dr. Hughlings Jackson's observations, and with those of Hammond and Durham. Mr. Durham also trepanned the skull of a dog, under chloroform, and watched the vessels of the pia mater. So long as the chloroform was being applied the veins of the membrane were distended and overfilled; when the dog fell asleep, the membranes became pale, the vessels fading and contracting. On arousing the animal, the surface of the brain again reddened, and pressed through the opening.

Hyperæmia of the retinal vessels may be a sign of general hyperæmia of the encephalic vessels, or it may be a sign of some obstruction affecting the ophthalmic veins wholly or chiefly. In the former class we must place the hyperæmia which is said to be due to some narcotics. It seems very probable that narcotics proper produce their effects by a venous hyperæmia of the brain; that the retinal vessels are very heavily congested during states of sopor and coma is, I think, made clear. M. Bouchut speaks in the most certain way of having witnessed this kind of action in the retinal vessels. In many cases he watched the action of chloroform on the retina during the inhalation of the drug, and in all he found a very marked injection of the fundus, with increase in the number and size of the veins. In some cases he noticed a capillary congestion and effusion masking the whole of the papilla and throwing a veil over it.

In one case the effects were so marked that M. Cuinier, an experienced observer, could scarcely believe in the reality of what he saw. In the '*British Medical Journal*' for May 27, 1871, a death from chloroform is recorded. After death,

⁸ It is not important here to distinguish whether the contraction of the vessels be due to antecedent diminution of the activity of cerebral tissue, or whether the latter be due to a contraction of the vessels.

Mr. Couper found the retinal veins greatly distended, but whether Mr. Couper considered this as a common sign or as a fatal sign, the deponent says not. Bouchut noted somewhat different effects on the administration of belladonna and opium. In these latter cases the capillary circulation is little affected, the disk seeming unchanged, while the veins are distended. This is very like what Dr. Jackson saw in natural sleep,—small arteries, that is, with large veins; so that opium sleep may be, perhaps, genuine anæmic sleep, and not the sleep of capillary congestion. In a paper in the ‘Practitioner’ (January 1871, p. 15) Mr. Goodhart states that he noticed the same change in the vessels of the retina on the administration of nitrite of amyl, the arteries, that is, were diminished in size, while the veins became dilated and varicose. Richardson has noticed the same thing, also, in the capillaries of the frog’s foot. I have neither had the time nor favourable occasions for the repetition of these experiments; when taken up, they must be carried out very thoroughly, and the results of the administration of many drugs noted and compared with each other, with the effects of natural sleep, and with states of coma from disease. However, my friend and former pupil, Mr. Aldridge, now one of the resident medical officers of the Wakefield Asylum, has taken up this subject with considerable industry, and I am enabled to quote the following conclusions from a paper which he is now publishing in the first volume of the West Riding Asylum ‘Medical Reports’ for 1871. Mr. Aldridge has investigated the effects upon the retina of the following drugs:—

‘1. *Potassium bromide*.—The effect on the circulation of the epileptic was not seen for some two months, and then a marked change was noticed in the calibre of the veins. Where they had formerly been dilated they became much reduced in size, and the capillary tint of the disk lessened; there was no change in the arteries. In fact, the state of passive hyperæmia which previously existed had become changed, and a more normal and active condition of the circulation induced. From these facts I concluded: “It would appear that the reduction in the amount of hyperæmia is not

induced rapidly as a direct effect of the action of the bromide on the blood-vessels, but is rather due to the diminution of the fits which give rise to the constant congestions."

'2. *Ergot*.—Was used in cases of epileptic mania⁹. The condition of the retinal circulation before its administration was one of great and active hyperæmia¹⁰. The drug did not cause any immediate and marked change, but in a few days or a week almost all signs of active congestion had subsided. In the same cases when the medicine had not been given the state of mania had lasted for several weeks, and the congestion of the retina had continued for the same length of time.

'I also gave the ergot to an old epileptic who had never been treated before. In the first instance, before the medicine was given, there was great passive congestion of the retinal circulation. In twenty-four hours there was a slight change observable in the arteries, their calibre having become reduced, and at the end of a week, although no change was observed in the arteries, the capillary tint had become paler, and the veins were not so dilated; at the same time, the fits had become reduced considerably in number.

'3. *Chloral*.—Twenty-five grains were given to a patient who had never taken the drug before. It produced no apparent effect either in her general condition or in her retinal circulation, which was examined every hour for six hours after the drug had been taken. In another case, thirty grains were given to an epileptic who was slightly excited, and whose retinal circulation was abnormally active. In one hour no effect, either general or local, could be observed; she then took a second thirty grains, and soon afterwards became quiet, drunk, and sleepy. At this period a very slight reduction in the size of the arteries was observed, and the disk was slightly paler. She was now put to bed, and in about an hour, when she was in a very sound sleep, the retinas were examined. The arteries had become slightly contracted, and the disk was paler than it had ever been. She was roused up

⁹ *Vide* article by Dr. Crichton Browne in the 'Practitioner,' June, 1871.—T. C. A.

¹⁰ *Vide* p. 83 of this volume.—T. C. A.

by walking about the room, and again examined. The tint of the disk had deepened, and the whole circulation had become more active. In two or three cases the same conditions were observed after taking the chloral hydrate. (*Vide* "Observations" of Dr. Hughlings Jackson, p. 273.)

'4. *Nitrite of amyl*.—A large number of cases were examined, and the results were found to be the same in almost every case. At the period of deepest flushing of the face and neck, the retinal circulation was found to be in a state of active hyperæmia, the arteries being notably increased in size. This appearance was, however, of short duration, lasting about the same length of time as the flushing of the face.

'5. *Nitrous oxide gas*.—The effects produced upon the retinal vessels by this drug resembled closely those produced by the nitrite of amyl, but were of rather longer duration. The patients were examined during the stage of greatest flushing of the face. In Dr. Mitchell's own person the effects were best seen, as the disk became of such a deep red colour as to be almost indistinguishable from the surrounding choroid. I was unable to get a view of the retina at the time when the face had a pale, livid appearance.

'I should state that the cases where chloral was given did not manifest the flushing which is sometimes seen to follow the exhibition of the drug.'

I have only to add a hope that Mr. Aldridge will pursue these most interesting and important investigations.

CHAPTER XIII.

THE EFFECTS OF MENSTRUAL DISORDERS UPON THE OPTIC NERVES.

It seems scarcely doubtful that women present themselves at eye-hospitals with disorders of vision which appear to be attributable to menstrual disorders. On the other hand, I have examined women innumerable, amenorrhagic, dysmenorrhagic, and hypermenorrhagic, until it seemed to me a mere waste of time to look farther, and yet without discovering any such coincidences.

Pagenstecher¹ speaks very strongly in favour of a connection between blood losses and neuritis or atrophy. He says, that in many cases and during a long period he has had occasion to diagnose changes in the retina as due to piles, and also to 'serious disorders' of the menstrual function. Three times he has seen atrophy of the optic disk supervene after menstrual disorders of long standing. In one of his cases, neuro-retinitis descendens is attributed to hæmorrhoids. For my own part, I feel great hesitation in accepting the assertion of a causal connection between such events. I have certainly found anæmia, and very marked anæmia, of the disks in such cases; moreover, in extreme anæmia, as I have said, we often find slight œdema also, but I have never seen neuritis or atrophy. I am far from hinting that

¹ 'Klinische Beobachtungen,' 3 Heft, Wiesbaden, 1866, pp. 67 and 75. Mooren also takes a similar view of the causation of many cases of neuro-retinitis, 'Beobachtungen,' pp. 294-297.

Dr. Pagenstecher has mistaken anæmia for atrophy; he is too skilful an observer to admit any such source of error; but I do mean to say, that cases of atrophy of the disk and of neuritis often crop up at ophthalmic hospitals, in which no such cause exists, nor any other cause which can be described, and I think it very likely that some of these may be credited to hæmorrhoids or menstrual losses, when the latter are really independent coincidences.

A very interesting case went the round of the foreign journals about two years ago, in which hæmorrhagic neuro-retinitis followed hæmatemesis. It is probable, however, that the two events were the common consequents of some strange affection in the medulla and upper cord. The lady was seized at first with an agonizing pain at the back of the neck, and the hæmatemesis and retinal mischief succeeded it.

That, as a general truth, inanition or large losses of blood enfeeble vision as they enfeeble hearing and touch, is scarcely worth asserting; and this debility seems to follow protracted suckling more commonly than any other drain. Many women have feeble vision, or even loss of vision, after an unusual drain of this kind. Deficient supply of blood from the heart is probably one cause, for in these women we find the heart pumping away with a great pretence of vigour, but making little effect upon the distal arteries. The præcordial movement is perhaps the labour of a distended right ventricle, the left ventricle wanting proper blood-supply; or it may be that the left ventricle, like the carotids, loses tone in such cases, and throbs painfully: when this occurs, in connection with enfeebled or capricious vision, the case is called one of amaurosis from congestion! The periodicity of nerve changes is well shown in states of optic anæmia from drain, the nerves at frequent times going quite to sleep, the patient falling blind for awhile.

Here I may refer also to those other cases, in which 'amaurosis' has been credited to worms (*vide* Appendix, cases 7 and 12), to the suppression of eruptions (*vide* case 25), to excessive or diminished secretions, and the like. These cases are generally recorded in a curious, rather than in a scientific

temper, and, like ghosts at cock-crow, seem to show a remarkable tendency to evanescence when submitted to the light of the ophthalmoscope. They mostly belong to the category of perverted sensations, or perverted perceptions, which are common in all parts of the nervous system, and are themselves of but little moment.

CHAPTER XIV.

EMBOLISM OF THE CENTRAL ARTERY OF THE RETINA AND ITS BRANCHES¹.

IN the first instance, I had not intended to include this subject among those treated in this volume; but, on subsequent thought, I venture to do so. Embolism of the artery of the retina is indeed a purely local event, and has its origin in an accident, rather than in any special constitutional state. It is a process, however, which has interested me very much as a physician; and I think, therefore, that it will interest my brethren, as being a specimen, and a visible specimen, of an event which frequently occurs in organs under medical charge. To see this process, and thus to be enabled to reason upon it when it occurs invisibly, is very valuable help. I shall therefore describe what is seen of arterial embolism in the eye, and shall make some reflections upon its phenomena, which have, I think, bearings of a general and important kind. The discovery and examination of the phenomena of embolism were made about sixteen years ago, and, although much was done to illustrate the discovery by our own Kirkes and others, yet the discovery itself is, I believe, to be attributed to Virchow². The first observer who minutely described the ophthalmoscopic appearances in the eye in such

¹ Liebreich, 'Atlas,' plate viii. figs. 4 and 5.

² *Vide* 'Gesammelte Abhandlungen,' 1856, pp. 539 and 711, where two examples of embolism in the eye are recorded. *Vide* also his 'Archives,' vol. ix. Pt. ii. p. 307, and x. Pt. ii. p. 179, 1856.

cases, was the great Von Gräfe, whose first memoir appeared in the Archives of Ophthalmoscopy for 1859. Liebreich soon followed with an account of numerous cases, and he has now personally observed no less than sixteen cases. Dr. Knapp, of New York, has lately collected all the cases of embolism of the vessels of the eye which are on record, and has added a very important case of his own, to which I shall have to make especial reference. No one has better described the phenomena than has Von Gräfe in his first case (*loc. cit.*), and I shall therefore use his own words in description. I have myself met with three cases of the kind, one of which was shown to me by Mr. Teale some years ago. As might be expected, there is great uniformity in the phenomena of the cases on record.

On Dec. 17, 1858, a patient presented himself before Gräfe who had suddenly lost the sight of the right eye a week before, at which time it was ascertained that he was suffering from traumatic endocarditis. While at work he perceived a cloud to form before his right eye, giving misty outlines to all objects. The field of vision then rapidly contracted, and in a few minutes the perception even of light was quite gone. On examination it was found that the right eye was absolutely blind, and that its iris only contracted sympathetically with that of the other eye. On applying the mirror the media were seen to be quite transparent, but the disk was very pale and its vessels reduced to a minimum. The principal arterial branches beyond the disk were also like fine lines upon the retina, the farther branches having quite disappeared. The state of the veins differed from that of the arteries, smaller indeed they were than normal in all places, but towards the equator they increased in size. The left eye was normal in every respect. Now this was not atrophy of the disk, for in no form of atrophy have we such a relation between veins and arteries; and, moreover, the papilla though pale was quite transparent, and not diminished in volume.

Gräfe then distinguishes between the inferences of an obstacle inside the retinal artery and outside of it, and again

of a rent of the artery, deciding in favour of the first hypothesis, which was subsequently confirmed by an autopsy. He also points out that the central artery must be the seat of the plug, as if it were above that artery there would be some interference with the choroidal circulation, which was not the case. When seen a week later, the region of the yellow spot was no longer normal. The central part of the retina began to veil the tint of the underlying choroid, and this veil soon became more evident as a distinct greyish white opaque infiltration. The foramen centrale appeared, on the other hand, to be surrounded by a patch of deep cherry-red; it was about one-quarter the size of the disk, and was placed in the midst of the infiltration. This intense red colour was shown subsequently by Liebreich not to be hæmorrhagic, but to be an illusive effect of contrast. On examining the infiltrated parts in large images it was found not to be uniform, but to present a number of whitish points, which, to judge from other investigations, would seem to be aggregations of fatty granules. Ultimately, some perception of light returned in the eye, as a little collateral circulation was perhaps established; but the great part of the affected retina, with the disk, underwent fatty degeneration.

Such were the phenomena in the classical case, and there is little to add to them. It will be seen in all such cases, as we should expect from the blood-supply of the retina, that this once cut off, there is little hope of any valuable collateral circulation being established. If we compare this case of occlusion of the main stem of the *arteria centralis* with Dr. Knapp's case, in which one intraocular branch only was affected, we find that in it blindness and opaque infiltration also occupy the part affected, but the veins of the same part are enlarged and tortuous.

There were also in Knapp's case numerous apoplectic spots in the affected region, chiefly around small venous twigs, or on both sides of the larger ones. Another very remarkable peculiarity in Knapp's case is the recovery of the normal calibre in the distal parts of the occluded artery. Dr. Knapp accounts for this by supposing 'that the sudden increase in

calibre of the retinal artery may fairly be accounted for as the place of junction of a retinal and a ciliary artery ;' and he adds, 'the blood seems to have been driven out of the arteries by the contractility of their walls.' Now this brings me to a point upon which I would lay some stress, as it is of great interest and importance in estimating the effects of arterial embolism elsewhere. The point is this, that the venous distension and the hæmorrhages are probably rather due in part to the action of the heart, which falls with a stress upon the open vessels, which is excessive in proportion to the lessened area over which its action is distributed. Secondly, that the fulness of the distal branches of the artery and the distension of the veins associated with it are due directly to a palsy of that extent of the walls of the occluded artery which lies beyond the plug. This latter fact, which has bearings of a most important kind upon the phenomena of ligature, and I think of embolism also, was established some years ago by Brown-Séquard, whose observations have not in this instance attracted the attention they deserve. In embolism of the *arteria centralis retinæ*, the opportunities for collateral re-establishment of blood-supply are too slight to enable us to reason exactly from it to other arterial regions more fortunately placed in this respect ; but the phenomena of the plugging of one branch of it, as described by Dr. Knapp, give us exactly what we want in order to reason, let us say, to the phenomena of embolism of the Silvian artery ; in which case we have also one branch of a system blocked up, and that one a branch which is but tardily supplied, as the branch in Knapp's case was tardily supplied, by associated but distinct members of the same system. Brown-Séquard's observations were made by him only in the case of ligature. He says, in a recent communication³, that ligature of an artery necessarily paralyses the vasal nerves which course along it, and consequently causes flaccidity of the distal portions of the vessel tied. The heart's impulse remaining the same, the

³ 'Arch. de Physiologie Norm. et Path.' No. 4, 1870, pp. 318, 319 ; where he refers also to statements by Prompt and Moreau, 'Comptes Rendus de la Soc. de Biologie,' 1868, p. 233, which I also have before me.

blood is forced accordingly into this district of least resistance, there being ever a correlative venous reflux in cases where direct anastomatic supply is difficult. He found even when he cut off all arterial supply to an organ (such as the kidney), save one vessel, that on tying this vessel the organ became congested by venous reflux. I need not say how direct an application these facts have in cases of ligature, say of the carotids, and how they illustrate the previously obscure facts of hyperæmia in parts beyond ligature. For instance, I was reading but the other day an interesting account, by Mr. Chatto, of a paper by Prof. Hueter on Arterial Transfusion⁴, in which the syringe was ligatured into the posterior tibial artery. Prof. Hueter remarks, that on transfusion, although extravasation has not been observed, yet the fine vessels undergo great expansion, the papillary bodies being filled with more blood than even in a condition of inflammation.

Now, if embolism has anything like the same effect which ligature has upon the distal branches of an artery, we have at hand a very interesting explanation of certain well-known and hitherto puzzling phenomena of these cases; such, for example, as the congestion, more or less venous, of the parts beyond the plug, and the strong tendency to hæmorrhage. It has occurred to me that the gangrenous character which supervenes in the inflammation of parts, such as the penis, where the inflammation causes plugging of main arteries, may be due to this kind of vaso-motor paresis⁵. A plug inside an artery is not, of course, so efficient a paralysing of its associated vasal nerves as a ligature would be; but the distensile force stretching the arterial coat behind an obstruction suddenly formed, is likely, as it seems to me, to have a similar, if not a co-extensive, effect.

In this volume I have to deal with the lights which the ophthalmoscope throws upon nervous disorders; and in the present chapter, therefore, I have to consider what light this

⁴ 'British and Foreign Med. Chir. Review,' July, 1870, p. 272.

⁵ And this would explain the success which has followed the practice of my colleague, Mr. S. Hey, who unloads the vessels in these cases by free local bleeding.

instrument throws upon that sadly common accident—embolism of the Sylvian artery. I considered this question somewhat fully in an article in the ‘Medical Times and Gazette’ of the 30th of April, 1870; and as I have little to add to what I then said, I shall reprint that article here. It will be seen from the opening remarks, that I applied the ophthalmoscope to test certain statements of Prof. Niemeyer, which had appeared in the same journal a few weeks before.

*On the Symptoms of Embolism of the Sylvian Artery, and
their Causes.*

I feel there is some presumption in my coming forward to discuss any points of Professor Niemeyer’s lecture; my only excuse is, that these cases of Sylvian embolism have for some years been a favourite study of my own, and that I have certain definite opinions concerning the phenomena of them, which opinions are somewhat different from those of Professor Niemeyer⁶. I hope I am justified, therefore, in taking the present occasion for a brief mention of some of them. At the same time let me express my sense of the great value and importance of Professor Niemeyer’s lecture, and let me particularly refer to his account of the very interesting collateral phenomena of splenic embolism in the case which he records. I do not think we in England inquire sufficiently into any history of pain in this region, or of shiverings, or that we look carefully enough for enlargement of the spleen with or without tenderness. His observations, again, on the discovery of encephalic embolism and thrombosis as pathological facts are very interesting, as they are in direct refutation of such strictures as that of Nélaton, which has attracted so much attention of late. I mean the absurd accusation against ‘mikro-

⁶ While these sheets were in the press we had the sad news, first of Gräfe and afterwards of Niemeyer, that their work was done. My first impulse was to strike out the controversial parts of the following paragraphs, but on second thoughts it seemed best to leave them as they stand. By none could it be more proudly said, ‘Non omnis moriar,’ and for us the greatest part of the master still lives.

scopische Spielereien,' which, according to Nélaton and others, have the tendency to encourage a beetle-eyed method, and to disintegrate all large conceptions of clinical phenomena. In encephalic embolism we have a discovery which was bred by this new spirit of minute research, which, however, is not a microscopical discovery at all, but one which was open to the naked pathological eye, and which was discovered less by poring among the tissues than by the suggestions of clinical phenomena themselves. So that in this, as in hundreds of like instances, the microscope is the companion of accurate and discriminating clinical observation, and not of petty curiosity.

The point to which I wish rather to refer to-day is that of the phenomena of consciousness and their causation. In the case of Sylvian embolism, which Professor Niemeyer records, unconsciousness was a marked symptom, and he speaks of unconsciousness as a common symptom of this disorder. Indeed, he refines upon the diagnosis between embolism and hæmorrhage, and considers that the loss of consciousness in both is one of the resemblances which tend to confound the two kinds of mischief; so that the ultimate diagnosis is rather to be inferred from the less direct evidence of the patient's age and the condition of the heart. In his subsequent remarks upon the pathology of the affection, Professor Niemeyer offers an explanation of this unconsciousness, and he refers it to an œdema of the parts which suffer—to an œdema, that is, of the mesocephalic ganglia and their neighbourhood, which presses upon the cerebrum. This is a very important question, and it is one upon which the diagnosis of Sylvian embolism more or less depends.

To take the symptoms first: it is certainly contrary to my experience of cases carefully watched and investigated to suppose that unconsciousness, or what I may call mental or cerebral apoplexy, occurs in them to any marked degree. These cases of embolism are not uncommon either in our waiting-rooms or in our museums, and I have generally six or eight under my own care among hospital patients and elsewhere. Now it is a remarkable and important fact, that

in my own cases I rarely get a history of complete unconsciousness. I very rarely find, that is, a history of coma or semi-coma, unless it be of very short duration. In many well-marked cases with complete or almost complete hemiplegia, I have satisfied myself, after careful inquiry, that there has been no actual unconsciousness at all, but rather shock and bewilderment. To speak pathologically, there has been in such cases no evidence of more than momentary pressure. For example, on turning to my notes, I find that a young woman suffering now from marked and obstinate hemiplegia, and from all the symptoms of left Silvan embolism and from heart mischief, never really lost consciousness at all. She had risen from bed but a few minutes or moments, when she fell, and was raised up hemiplegic; she was 'dazed,' but remembered indistinctly what had passed, how her friends came about her, how her husband lifted her, and how she woke from a sort of confusion to find her right side useless and her speech impaired. To take a second case: a woman was engaged in household work, 'a sickness and fainting came over her,' but she was able to reach her chair; she seems then to have been 'lost' for a few seconds, from her daughter's account, but soon recovered consciousness, remaining, however, hemiplegic of the right side. Another woman was standing on a chair to reach something above her, when she fell to the floor hemiplegic⁷. Again, Mrs. H., about 40 years old, and of healthy appearance, had rheumatic fever seven years ago, and has now heart mischief. Nine months ago, on rising from her breakfast-table, felt bewildered and fell. Unfortunately, she was at the moment alone. She arose again, feeling in a dream, and again fell. Three times she essayed to stand and fell, but on the third occasion she managed to fall into a chair, and ring the bell. She is sure she never lost consciousness, but was bewildered and 'dazed.' When her friends came in, they found her quite hemiplegic on the right side, and also speechless, or nearly so, but not unconscious. She is still severely paralysed on the right side, and talks with much hesitation.

⁷ Embolism often occurs after or during some effort, or during an attack of palpitation.

There was but transient unconsciousness. The same stories I find also in the histories of male patients attacked with left Silvan embolism. I am told of their being 'dumb-founded,' 'faint,' 'lost for a few minutes,' and the like, but seldom really comatose or stertorous. I lately saw a very curious case of this kind near Manchester, in consultation with Dr. Eason Wilkinson and Mr. Mellor. A healthy young man, having no syphilitic antecedents, went down to his office in Manchester, and while there made many strange and inappropriate entries in his books. On returning home to dinner in the middle of the day, he was observed to be twitching his face, especially on the right side, and to speak indistinctly. During dinner, also, he made grimaces, and was rather confused in mind and speech. He also often let fall his knife from his right hand, so as to attract attention. On starting to return to Manchester, he became unable to hold his umbrella in his right hand; his leg also gave way, and he was brought into the house again, completely palsied on the right side, and also completely or almost completely aphasic. In this state I saw him. We all agreed, without any ultimate ground for doubt, that this was a case of embolism, in which the plug was at first but partially impacted, and probably acting for a few hours as a ball valve in the arterial pipe. Here we are supported by the evidence of the ophthalmoscope, which proves that some hours have elapsed in some cases before the plug became fixed, before the arteries were quite occluded, and before vision was wholly lost. In all these cases, I refer, of course, to embolisms of the main artery with grave persistent hemiplegia, and some speechlessness; not to embolism of smaller branches, on the one hand, or of the internal carotids or anterior cerebral arteries on the other hand. In all cases of simple Silvan embolism, loss of consciousness seems to me to be far from a prominent symptom. Still, in some cases, it does occur; but these cases form a small minority of the whole: in them, I believe, there is some collateral hæmorrhage in addition to the embolism⁸. Now, this absence

⁸ Embolism may, of course, be followed soon by 'red softening' in the hemisphere, when there may be serious disorder of consciousness.

or slighness of the unconsciousness is very remarkable when we compare it with a hæmorrhage of like extent. A hæmorrhage into the corpus striatum sufficient to reach the speech districts, and injuring the corpus itself so far as to produce grave and abiding hemiplegia, is always attended with decided, if not with serious, coma, with coma lasting, perhaps, for several hours, and accompanied with more or less stertor.

So far for the symptomatology: let us now inquire into the pathology of the affection. Embolism occurs in one place where we can see it—namely, in the central artery of the retina; and I have been in the habit of collecting all the observations I can find of this disorder as illustrative of the same process in the encephalon⁹. I have also had the opportunity of seeing three cases—one which was shown me by Mr. Teale, another which I watched myself for a long period, and a third, which, however, was of somewhat doubtful origin, but probably embolic. Now, the phenomena in these cases, if the plugging be fully established at once, are—

1. Instantaneous, or almost instantaneous, loss of function.
2. Sudden emptiness of the arteries and capillaries, and more or less of the veins.
3. Œdema of the parts deprived of supply, which œdema comes on gradually, and, in cases seen early, amounts to little more than a slight haziness around the yellow spot.
4. A tendency in the later stages to hæmorrhages from collateral vessels (peripapillary and choroidal).
5. Ultimate thickening of the adventitia of the vessels and fatty degeneration of the retinal tissue, with deposit of cholestearine (Schweigger, Augenspiegel).

I think we may fairly assume that the changes in the encephalon are of the same kind—namely, sudden emptiness of the arteries and corresponding venules, with some momentary deficiency in the contents of the sinuses, œdema of the affected parts, tendency to collateral hæmorrhages, and instantaneous loss of function. One condition, however, which we have not in cases of embolism of the main stem of the

⁹ Compare especially Liebreich, 'Deutsche Klinik,' 1862, No. 50; his 'Atlas,' *loc. cit.*, and previous pages of this chapter.

arteria centralis retinæ, but which we have in Silvian embolism, and which we have to some extent and may see in the embolism of a secondary retinal artery, as in the case of Dr. Knapp already mentioned—is this, that there is a possibility of some supplementary circulation. Though this, even in the Silvian district, is far less than, say, in the district of the mesenteric artery, yet it must have some effect, and an effect of the kind I have anticipated on a previous page. We shall have, as in Dr. Knapp's case of the retina, quick filling of veins and of the distal branches of the Silvian artery with blue or bluish blood; and we may also have, as in Dr. Knapp's case, a number of small hæmorrhages.

Now, such being the pathological conditions, how are they related to the symptoms? Instantaneous loss of function of course is itself a symptom, and I merely mention it in the present connection, because we are enabled to see from the changes in the eye, which we can follow, that the loss of function is immediate. The promptness and severity of subsequent hæmorrhages seem to depend upon the strain on collateral vessels, and on the degree of the vasal paresis. For example, in embolism of the mesenteric arteries, I believe, abundant hæmorrhage always occurs. In the eye, on the contrary, where collateral vessels are few and small, hæmorrhages are few and small. In the encephalon we more frequently find a tendency to hæmorrhage; and hæmorrhage, when it occurs, produces by its pressure anæmia and loss of function in neighbouring parts, over and above loss of function in the parts directly cut off from the circulation. There was this tendency to hæmorrhage in the case recorded by Professor Niemeyer. Lastly, as to the œdema. To this, Professor Niemeyer attributes the unconsciousness, supposing that it is the agent of pressure upon the hemisphere, or brain proper. Now, a study of embolism in the retinal artery shows us that the œdema, so far from being immediate, creeps on somewhat slowly¹⁰, and that a patient seen soon after the accident presents but little œdema. This œdema occurs, then, not as a

¹⁰ 'Einige Stunden' (Blessig), or, 'am folgenden Tage nach Beginn der Erkrankung' (Liebreich).

'serous apoplexy,' but as a gradual infiltration or soaking of the tissues, which is in some way consequent upon the emptiness and paresis of their vessels. The œdema may also be partly due to a change in diastatic relations or in vascular tone; but, I think, in the encephalon it is chiefly compensatory, and is to be likened to the emphysema which compensates atelectasis in the lung. An arterial region is emptied, collapse follows, and we sometimes find, in addition to local œdema, an excess of fluid, which may be blood-tinged, in the subarachnoid spaces or in the ventricles, with or without an evident accumulation of venous blood in neighbouring parts. The greater degree of exudation is not seen, I think, in embolism of the lesser vessels, but is recorded in embolism of the carotids.

œdema then is, I believe, a process subsequent to the plugging, and is not a cause of pressure upon surrounding parts, but is rather a support to parts which are in danger of collapse. Hæmorrhage, again, is, in the eye, a late process, and never simultaneous with the embolism. I believe the same is true, or almost true, also of the encephalon. Hæmorrhage, therefore, is not often a prompt cause of pressure upon the hemispheres in these cases; so that both clinical and pathological experience lead me to deny that unconsciousness is a grave symptom in Silvian embolism, and to maintain that in this the symptoms of Silvian embolism differ from those of encephalic hæmorrhage in the same region and of equal extent.

I admit that, although in some cases of Silvian embolism there is no unconsciousness at all, unless a transient bewilderment be called unconsciousness, yet in many cases there is no doubt an interval of unconsciousness, though far less than in a case of hæmorrhage of equal extent. What is the cause of this transient unconsciousness? Now, as this symptom is sometimes absent, and, if present, is present in very various degrees, there are probably two or more causes at work which may alone or together throw the action of the hemispheres or cerebrum proper into temporary abeyance, the caprice of the symptom being probably due to some want of uniformity in its causation. I believe that unconsciousness may be due to one or all of the following causes:—

1. That degree of venous reflux and fulness of the distal vessels which we have seen in the eye in Dr. Knapp's case, and to some extent in other cases also, and which is due to a certain degree of vasal paresis caused by the local pressure of the plug.

2. That shock, which in the whole encephalon seems by a certain sympathy, as it were, to echo the shock of sudden injury to any one of its principal parts. It is a matter of common experience in medicine, and likewise in experimental pathology, that partial injuries, if slowly induced, may be unfelt by neighbouring and connected parts; but that, if suddenly induced, they are followed by great disturbance of such other parts. This disturbance is shown in losses of sensibility, in rotatory movements, in vertigo, and the like. In this way, it seems to me that the sudden collapse of the mesocephalon in Silvian embolism may be echoed in the hemispheres, and thus give rise in them to some transient molecular disturbance and some transient change in corresponding function. It seems certain that the sudden emptying of important blood-vessels cannot take place without collapse in the parts cut off, and dislocation in the parts which are near them.

3. There must be a moment of increased tension in all the encephalic arteries at the time of the plugging of the Silvian artery. The next heart-stroke and the next systole of the carotids is expended upon a smaller extent of surface than before. In cases where the vessels are diseased, prompt hæmorrhage might occur in this way, and would occur rather in parts whose normal resistance is lessened—in the parts, that is, which surround the Silvian region, and which, by the collapse of this region, are deprived of their normal support. But it is merely to the increased fulness of the arteries in the hemisphere, and in the membranes above and beyond the Silvian region, to which I would especially refer as a consequence of the temporary increase of strain, and as likely to produce a momentary effect of concussion when the extra blood is thrown into them from the carotid, whose calibre remains unchanged.

CONCLUSION.

AND now, kind reader, I have done, and it only remains for me to conclude, gracefully if I can, but in whatsoever way—to conclude. I say kind reader, but I much fear the kind reader is dead; I fear it, often wondering and hoping that he may still live—that courteous gentleman who bought our books, read every line of us, welcoming our truths, ever kind to our faults, and pulling off his hat if he thought it well to correct us. For his own sake, indeed, I sometimes trust that he is gathered to his rest amid the eternal tranquillities of his more and more gentle and learned forefathers. If he now live, his courteous forbearance must be greatly exercised by us modern scribblers, and his fine ways must be grievously hustled in this growing world. Yet, if he be dead, I sadly think, then there is none to read these lines, for none else would bestow himself so far upon me; none else would set me so far upon my journey; there is no other, and I will write this silent memorial of him. Should he, however, be still abroad—sometimes, perhaps, of fine summer evenings—then, kind reader, I thank you heartily, and trust, indeed, I may have contributed somewhat to your pleasure and your profit; to your pleasure more, to your profit less. For while I would not be vain enough to think to add to that learning and wisdom in which you already abound, yet to your generous mind I may hope I have offered that which is most pleasant to it, namely, work done in an earnest belief that no facts are small, no careful testimony trivial; that the happiest pursuit in human life is to seek out its truths, and to learn its ways; the greatest honour to have helped, be it ever so little, to promote that science which ‘should be a rich storehouse for the glory of God and the relief of man’s estate.’

FINIS.

APPENDIX.

CASES.

The Cases printed in the Appendix are intended to have an illustrative value only. In them brevity and certainty seemed to the author to be of the first importance; and it is hoped that the notes given are sufficient to identify the conditions observed. Few cases depending upon an autopsy are included unless an autopsy was obtained. The author has to regret that some of the most curious and interesting cases in his ophthalmic collections must, for this reason, be omitted. Many other cases are also omitted which add nothing to our knowledge, or which only add, without important difference, to the illustrations here given.

A P P E N D I X.

C A S E S.

1. *Epilepsy preceded by temporary blindness.*

F. P —, æt. 17, was in health six months ago, when she was seized by a fit, which has recurred sometimes twice or thrice a week; sometimes, however, not even once a week. Generally they occur about noon, but sometimes later. She struggled a good deal at first, but less so of late. At present, Sept. 14, 1868, the first symptom is loss of sight, but this has only been the case lately; previously, she had no warning. There is complete darkness and no coloured vision. In about two minutes she falls, and thence recollects no more. She lies pretty quiet, and is insensible from half an hour to an hour. Latterly she has slept after the fits a good deal, and if so is much better. No palsy or other permanent symptom. (The last fit was two days ago.)

O. S. Disks seem perfectly healthy. Veins may be a trifle full.

If she does not sleep on recovery her sight remains weak for perhaps half an hour, after which sight quite recovers, and is now quite good.

2. *Two cases of epilepsy, with notes of the state of the disks after a paroxysm in each.*

‘The following are notes of an ophthalmoscopic examination made in two cases in which the patients complained of headache with a little dimness of sight *after a paroxysm* of epilepsy. My object was of course to learn somewhat as to the condition of the intra-

cranial circulation *after* an attack of epilepsy, and also *to see* the state of the vessels of the retina and the nervous tissue (the optic disk) supplied by them, in what, judging from the patients' statement, would generally be called 'congestive headache.' For, as the arteria centralis retinæ may be almost considered as part of the cerebral circulation, we shall no doubt learn, from studying its varying conditions, something as to the condition of the other branches of the internal carotid which supply the brain.

'In a case in which an epileptic complained of slight dimness of sight and headache after an attack of epilepsy which she had had the same day, the note is as follows:—

"She (a girl, æt. 8, idiopathic epilepsy, and tongue-biter) had a fit at 8 a.m. I examined by the ophthalmoscope at 10.30 a.m., without using atropine. I find the veins remarkably large and dark, and the arteries also seem darker than normal. The disk is hyperæmic."

'In another case in which after a fit there was dimness of sight and frontal headache, the veins appeared very large, and the optic disk was reddened as if flecked with red. It was not uniformly coloured, but somewhat like white bibulous paper slightly smeared by red ink.'

Hughlings Jackson, 'Med. Times and Gazette,' Oct. 3, 1863.

Also—

'A patient, William T., æt. 8, had been attending some time under the care of my colleague, Dr. Brown-Séquard, for epilepsy. One day the porter brought the boy (who had just been taken with a fit) into my room in his arms. Unfortunately he did not at once take him to the lamp, and, when everything was arranged for looking, the convulsions had, I think, ceased; but I was so much occupied in finding the optic disk, that I noticed little else. The pupil, however, was still very widely dilated, and I caught the optic disk of one eye. It was whiter than normal, and the veins were large and dark. I soon lost it, and then the pupil rapidly became so small that I could not again illuminate the fundus. It is of course of great moment to ascertain the exact stage of the paroxysm when the disk is seen.' *Ibid.*

3. *Epilepsy, hyperæmia of disks.*

Ann P——, æt. 18, was quite well three years ago, when she was terribly frightened at a pic-nic. The same day she was 'taken

in a fit.' Since that time fits have returned, often with great frequency. They are ordinary complete bilateral epileptic fits.

O. S. Examination. We examined the disks on one occasion (April 29, 1879) in the out-patient consulting room during the conclusion of a fit. Both disks were hyperæmic, especially the left disk. She was removed to Wakefield Asylum in a month after she commenced attendance.

4. *Epilepsy, retinal hyperæmia, disks normal.*

Miss H., a quiet, intelligent person of about 22, consulted the author for epilepsy. I need not describe the case, as the points of interest in it would lead us away from the present subject. The fits were rather frequent, appearing once or twice at menstrual periods. They were bilateral and rather severe. I do not think there was any 'coarse disease' in the brain. On examination with the mirror, within a few days of the attacks, the veins of the retina were seen to be enormously dilated and somewhat tortuous. Pulsations even in the smaller branches were distinct. There was probably great fulness of the cerebral sinuses. The disks were but little, if at all, hyperæmic. Mr. Teale twice saw this state of the vessels. The fulness of the retinal veins generally subsided in five or six days, to return again with the epilepsy. I never saw her immediately after a fit.

5. *Convulsions, hyperæmia papillæ, death, autopsy.*

Anne G., æt. 22, was admitted into the General Infirmary under the author on the 25th of February, 1868. She was a person of doubtful character, her history unknown, and the fact of her present seizure very difficult to obtain. It seemed, however, from the evidence of a man to whom she was talking at the time, that she had fallen in Briggate three days before in a state of convulsion. She had recovered, but the fits had returned before admission. She was in a semi-comatose state when brought in. Twenty-four hours later she passed into a 'status epilepticus' (coma and convulsion) which continued till death. Both sides were affected, and to the same degree: the iris was strongly convulsed. I examined her eyes repeatedly, and during long times, while both coma and convulsion was going on, as my assistants and the nurses were able to hold her tolerably still. I constantly found a fulness and slight tortuosity of the retinal vessels, a full redness of the disk, including the central

connective tissue, dim œdematous margins, and a large number of radiating vessels. I could see no neuro-retinal change during the convulsions of the iris. The patient died in about twenty hours, and, as I expected, we found only a marked fulness of the meningeal veins and of the sinuses, rather numerous puncta, some excess of serum in the ventricles, and a diminished consistency of the motor ganglia.

6. *Epilepsy, anæmia of disks and retinas.*

Jane W——, æt. 11, has suffered from bilateral ‘genuine’ epilepsy for eighteen months. The fits have recurred at uncertain but at somewhat short intervals. She was admitted into the Leeds Infirmary on May 1, 1868. I then found that the disks and retinas were quite normal in all respects.

On July 23. Has had two fits this morning about four hours ago and eight hours ago respectively. The retinal vessels are now fine in both eyes, the disks are pale, and the left disk is much paler than the right.

On subsequent examinations I noted that the disks were perfectly normal, the vessels being full and well marked rather than fine, and the disks reddish. The disks presented no difference of colour.

7. *Epileptiform seizures, aura from the thumb, attacks of coloured vision.*

(Under the care of Dr. Hughlings Jackson.)

‘Alice F., a married woman, æt. 49, was admitted an out-patient, under the care of Dr. Hughlings Jackson, on November 28, 1862. Until about seven or eight months ago she had had good health, and indeed looked still in fair health, and was very intelligent. She had not menstruated for twelve months, and complained a good deal of “sinking,” of faintness, weakness, &c.,—symptoms so common at the change of life. Until the attack to be described she had had no definite ill-health, except a pain in the right arm, of no very special character so far as could be ascertained.

‘Five weeks before she had a tingling sensation in the right thumb. It began under the nail, and extended about as high as the styloid process of the radius, and then “went to the face.” The part of the face first attacked was the upper lip on the right side; next, the whole of that side of the face, and the tongue also; “it took her

speech away for five minutes.” She was not at all insensible. She had great pain in the arm, but the leg was not affected in any way. She had had about twenty of these attacks before admission.

‘It is interesting to note that, before any question of any kind was asked, she said that catching the shuttle with the thumb and finger, and even touching the thumb, would sometimes bring on the fit. She gave a circumstantial account of this. She had also attacks in which there was coloured vision, which were distinct from the seizures just described. The first was about four days before the seizure described above.

‘This patient took iodide of potassium. At her next visit she was better, and had only had one attack. It was now ascertained that she had had tapeworms three years ago.

‘December 19.—She had had no fit, but “had had the colours dreadful.” It affected the right eye only, as she shut each eye in turn, in order to ascertain. The coloured vision was attended by pain in the right superciliary region. Both the pain and the colour came and went suddenly, lasting each time about ten minutes. She could see things in spite of this peculiarity, but rather dimly. The colours were violet, white, and orange, and seemed about three yards distant. At other times the sight was good, and she had no pain. She had not at any time during these attacks any vertigo or insensibility, but numbness of the right leg. She had considerable pain still from the elbow to the fingers, but this was constant. A dose of the oil of male fern was prescribed.’

‘Medical Times and Gazette,’ June 6, 1863.

I have had two or three cases of this kind under my care, but they do not differ importantly from this of Dr. Jackson.

8. *Attacks of unconsciousness and of dizziness, transient blindness.*

Mr. G. C., æt. 24, consulted the author in November 1868 for attacks of sudden unconsciousness, which often caused him to fall. Their duration was short. He had, however, suffered from almost constant dizziness for nearly two years. When he has a ‘full attack,’ he is blind for some minutes after his recovery. He is never convulsed. He has lost flesh, and has a worn and anxious aspect.

O. S. Disks very hyperæmic, and edges hazy. The vessels are full and dark. The left eye is the worse. I prescribed cod liver oil and oxide of zinc.

He gradually improved until Jan. 7, 1869, when he became again worse. Has had several attacks of unconsciousness, and much dizziness.

O. S. On this day I find the right disk much the worse. The left disk is hyperæmic and indistinct, but the borders are to be made out. The same treatment was continued, and the patient is recovered. On the 14th of March the disks were quite natural in appearance. All dizziness had disappeared.

9. *Periodic nervous disturbance, hyperæmia of disks.*

Mrs. S. R., æt. 23. Sixteen months ago had the first of a series of attacks, which have recurred at varying intervals up to March 4, 1870, when she came under the author's care.

The attacks could not be called epileptic in name, however near their affinity to epilepsy. Cramps and tremors seized the arm and leg of one side or of the other, or arrested speech; and when speech was arrested the thorax was also fixed and breathing stopped. Consciousness was never lost, though transient giddiness, bewilderment, and loss of vision would occur; sometimes she even fell, but retained perfect remembrance of all that passed. No marks of hysteria in symptoms or in constitution. Twice I saw her just at the end of an attack which had lasted ten minutes in the first instance and half-an-hour in the second instance. On both occasions the fundus was very red and injected, and slight œdema veiled the disks. In the intervals appearances normal.

10. *Epilepsy, transient blindness.*

'The total blindness preceding the epileptic paroxysm is a different thing; and just as epilepsy is supposed to depend on contraction of the vessels of the brain, so the temporary amaurosis, in these cases, probably depends on contraction of the blood-vessels of the retinae—an epilepsy of the retinae.

'I believe that the following is an instance of epilepsy of the retinae. It seems clear that it was not merely failure of accommodation. One morning Julia W., a middle-aged woman, came to me, saying that for five whole minutes she had been "blind." She was at the time seated peeling potatoes. The blindness came on suddenly and left suddenly. It was not total darkness, but "dark," which was the word she used herself in describing it. It was not from failure of accommodation. I asked her to look through a

very strong convex glass. It was not like that, she said. It was not spots, nor specks, nor clouds, nor colours. When I saw her a minute afterwards, she could read well with each eye, and the fundus of each, as seen by the ophthalmoscope, was normal. She had headache across the forehead, which continued the next day. She said it felt "tight" across the forehead. She had no giddiness. She was regular, but subject to dyspepsia.

'In cases in which loss of sight is followed by the epileptic paroxysm, may we not say that the contraction of the blood-vessels has begun in an outpost of the cerebral circulation (the retina being supplied by branches of the same vessels as the brain, these vessels being supplied by the same vasomotor nerves), and that, on extension to the other branches of the carotid, the "brain's blindness," loss of consciousness supervenes? In but one of the cases of disease of the cerebellum which I have reported, was there any note of temporary intermissions of sight. As a rule, the progress is gradual, and the loss of sight permanent.'

Hughlings Jackson, 'R. L. Oph. Hosp. Rep.' vol. iv. Part i.

11. *Periodic nervous disturbances, transient dimness of vision, eyes normal?*

Miss E. C., æt. 29. Came under the author's care early in 1870. Two years ago, when in ill health, became subject to attacks of dimness of vision not amounting to actual blindness. The dimness would, however, prevent her from distinguishing objects for about half an hour; it was accompanied by nausea, and often by vomiting. As it passed off it left a severe darting pain through the temples. These attacks troubled her almost daily for about two months, at certain times of the day; the least exertion being sufficient to bring them on. Lying down always relieved them. Her health has been feeble ever since, but no attacks recurred until a week ago, when they reappeared just as before.

O. S. I examined her eyes on one occasion twelve hours after a bad attack. The fundus seemed too red, and redder in one eye than in the other; but I could not feel at all sure that there was abnormal injection.

These cases are very common, and the optic changes may be significant of changes in encephalic vascularity.

12. *Morning blindness, tapeworm, anæmia of disks.*

Sarah H——, admitted into the Leeds Dispensary, Oct. 28, 1867, æt. 29. For twelve months has been subject to loss or dimness of sight every morning on rising. It generally lasts for about an hour and a half, and she is sometimes quite blind for that time. She never has the attacks at any other time. Under treatment she passed a great quantity of tapeworm, parts of which she had passed before. No great difference in the sight, however, could be noticed. She was then ordered a steel and tonic mixture, and in about three weeks she entirely lost the disturbance of vision.

O. S. On admission is noted, the vessels small and the disks 'slightly atrophic in appearance, but probably only pallid.' When discharged, the note of the disks is 'natural.'

13. *Headache, vertigo, hyperæmia papillæ.*

M. W., æt. 32. His sight had been imperfect for about a twelve-month, and on that account he came to Mr. Teale's clinic, where the author saw him. He complains of having suffered for some time from constant vertigo, occipital headache, debility and depression. Says that his habits are in every way steady, and he can give no reasons for his illness. The heart is healthy and the urine normal. Both disks are markedly hyperæmic, and the veins full. There are one or two twists or dilatations of vessels upon the disks, which, unless highly magnified, resemble hæmorrhages.

14. *General anæmia, vertigo, fiery flashes, anæmia of disks and retinas.*

Mrs. M., a patient of Mr. Mann's, was first seen by the author on April 13, 1868. She was a well-made and fairly nourished person, who had lately been confined, and was partially suckling her baby. Four years before, when in good health, had suffered a most painful shock, followed at the time by great prostration. She had since been profoundly anæmic, and was in this state at my visit. She complained much of palpitation, vertigo, numbness, and prickling in the limbs. The left arm and leg are chiefly affected, and we feared that there was some weakness of these limbs¹. Her most painful symptom, however, was the recurrence of fiery flashes in her eyes; flashes which were so sudden and so bright as to alarm

¹ *Vide* paper by the Author on Functional Hemiplegia, 'Br. Med. Journ.' Oct. 1, 1870.

her very much, accustomed as she was to them. They recurred like epileptic fits, at uncertain intervals, and generally at night. During the last year and a half she had suffered also from night blindness.

O. S. The back of the eyes are extremely anæmic, the vessels small, and the disks resembling grey white paper. There was not atrophy.

The result of treatment is interesting. Chalybeates and nervine tonics having been long and well tried, we agreed to order bromide of potassium in increasing doses, looking upon the flashings as epileptic. Our hopes were justified; the attacks were commanded by the drug, and with the addition of cod oil and other supporting treatment, she was so much better in eight or ten weeks that I took my leave. It seems probable that there was no local disease of the brain, although the suggestion of hemiplegia made us cautious in our opinions. She still remains in good health.

15. *Transient hemiplegia with aphasia, and blindness.*

Mr. B., a sensitive, intelligent man, engaged in laborious underpaid work, and suffering under needy circumstances and mental anxiety, is seized at various intervals by fits of the following kind. He becomes sick at the stomach, and dizzy; then his sight fails, leaving him altogether for about three minutes or less; his right arm and leg are enfeebled and very slightly convulsed, and speech is lost.

Before the absolute loss of speech he uses inappropriate words and feels much distressed at his inability to express himself. He is likewise unable to write anything coherent or to the point. Consciousness and intelligence to ingoing language are unaffected. The attack passes off in about ten minutes with free vomiting. His relief is then complete, and his memory unbroken. There seems to be no organic disease, and he improved while under the author's care with rest and tonic treatment.

O. S. The optic disks during the attacks are distinctly blanched, and the vessels fine. They do not recover their full calibre for twenty-four hours.

16. *Periodic nervous disturbance, transient amaurosis, fundus normal.*

Mrs. A. M., æt. 28. Admitted Leeds Infirmary, July 1870. Much pulled down by death of several children and some mis-

carriages. Nothing further suggestive of syphilis. Six months ago, when pregnant and feeble, but with no definite ailment, was seized with hemiopia while cooking. Was unable to see the left half of the pot, or of any object; then the tongue began to tremble violently, and the lips also. Then the tongue seemed to get too large for the mouth (anæsthesia?), and a numbness ran down the left half of the face and the left arm. In two hours sight returned, but the arm was 'numb' for twenty-four hours. A second attack occurred four days ago at 9.30 p.m. Suddenly objects became indistinct, then for a few minutes absolute darkness, then again indistinct vision and sparks. During the dark time the tongue 'again became much too large for her mouth,' and her lips became very numb, while a numbness also ran down to the end of the left fingers. On retiring to bed it passed off, but she had a threatening next morning again.

O. S. The fundus of both eyes was quite normal.

Vide paper by the author on Functional Hemiplegia of Women, in 'Brit. Med. Jour.' Oct. 1, 1870.

17. *Attacks of unconsciousness, congestion and œdema of disks.*

George C —, æt. 23, was admitted under my care in the Leeds Infirmary on Nov. 27, 1868. He is liable to attacks of unconsciousness, in which he falls to the ground. He has no struggling, nor ever had any. This we learn from his friends. He soon comes round, being seldom unconscious for more than a few seconds, but he remains blind for some minutes. As he cannot enter the house, we have no opportunity of seeing him in a fit, though they are frequent. There is no heart disease.

O. S. Examination on Nov. 27, twenty-four hours after last attack. Disks very hyperæmic, and edges hazy. Vessels full and dark. Left disk rather the worse.

O. S. Examination, Jan. 7, six hours after attack. Disks as before, except that the left disk is clearer, and the right disk worse, being now worse than left.

O. S. Examination, Jan. 14. No attack since the last mentioned, viz. Jan. 7. Disks both almost entirely cleared up.

Under tonic treatment with bromide of potassium the attacks were postponed, and finally prevented. After the attacks the disks were always congested, and cleared up in the intervals. On dismissal the disks had been normal for many weeks.

18. *Aortic regurgitation, anæmia of disks.*

Elisha W——, æt. 41, admitted under the author as out-patient on March 20, 1869. Ill some months. Aortic obstruction, and regurgitation in extreme degree. Liable to constant dizziness; often falls. At such times consciousness is seldom quite lost, but he seems in a dream. Speech will depart also for many minutes. Face and lips very bloodless.

O. S. Examination. Both disks white, equally affected, and like atrophy of the first degree. Retinal vessels too fine. Has often flashes of light in eyes, and mists. Field of vision complete.

Under steel and digitalis his general health much improved, and the disks became a little warmer in colour; but he ceased to attend after a few weeks, and before they had fully recovered their natural tint. The evidences of disordered cerebral circulation also diminished during the time of his attendance.

19. *Chorea, optic signs, recovery.*

‘On September 6 I looked at the optic disks of Caroline B., a girl ten years of age, and, as I was sitting down, I remarked to the students that I had never found any striking changes in the eyes of a choreal patient. I found them in this case. The changes were most in the right eye. The optic disk was badly margined; the veins were large and very irregular (wavy); and the disk was hyperæmic. There were similar, but slighter, appearances in the left eye. Now in this case, as in most cases of chorea, one side of the body (the right) was affected. It was the side only affected. There was, as I believe there always is in chorea, paralysis, and in this case the paralysis was considerable. The child’s talking was bad. It was, when I saw her, slightly thick. I took her into the hospital (thanks to the permission of my senior colleague, Dr. Davies). The irregular movements degenerated into paralysis, so that the arm was almost immoveable, and the changes in the optic disk increased. However, she ultimately regained much power, and the disks resumed nearly a normal appearance. She went to a Convalescent Institution in November. The arm was then nearly restored.’—Hughlings Jackson, ‘R. O. Hosp. Rep.’ v. p. 288.

20. *Meningitis, atrophy of disks.*

Esther E., æt. 15 weeks, came under the author’s care in October, 1868. Three months ago was seized by convulsions, and has had

many fits since then. Sometimes they consist only in a divergence of both eyeballs to the left: sometimes all four limbs and the face and neck are affected. There has been headache and vomiting throughout, and these symptoms seem to recur periodically, like the convulsions, and to disappear as suddenly. The head is natural in size, and there is no palsy, but the right side is generally more convulsed than the left.

O. S. Examination. Both disks very white, borders irregular and clouded, vessels not diminished. Child seems not to notice anything. Under treatment by iodide and bromide of potassium, with cod oil and steel wine, the child improved for some weeks; but at the end of the year she relapsed: fits, headache, and vomiting severe, the fits generally setting in fifteen minutes after sleep had commenced. The right arm and leg became palsied.

The child was an out-patient, but I learnt that she died on April 8, 1869. I was unable to obtain an autopsy.

21. *Meningitis, central softening, neuro-retinitis.*

A. E. O'H——, æt. 4 years, has been ill six months with headache, vomiting, and loss of flesh. A few slight fits have been noticed. She had begun to talk very nicely, but by degrees had now quite lost all speech, except a few baby words. (Loss of memory of language?). She was also losing power in all four limbs. Her hands are almost constantly clasping her forehead, and she screams at night. Muscles of orbit normal.

O. S. Examination on admission. Typical neuro-retinitis descendens, in both eyes. Vessels about normal in diameter, except where lost for more or less of their length in the reddish grey exudation in and around the disks.

The mother brought the child for three or four weeks, and then disappeared.

22. *Phthisis, meningitis, amaurosis.*

Harriette S., æt. $7\frac{1}{2}$, was admitted under the author's care into the Leeds Infirmary on Feb. 11, 1870. Mother is 'delicate,' and one sister died of consumption. When one year old, began to lose flesh and appetite, and to cough. Recovered to some extent under treatment, but is liable to a return of the symptoms every spring and fall. Six months ago was attacked with intense pain in head, constant nausea, and attacks of vomiting. Five months ago convulsions

appeared, and lasted more or less for fourteen days. At this time some failure of sight was noticed. After the fits she seemed to improve, and has slowly become convalescent, though she remains slightly hemiplegic of the right side, which side was chiefly affected in the fits. She is now quite blind.

O. S. Disks bright white, and vessels atrophied. There are marked signs of phthisis in the chest.

23. *Meningitis, ophthalmic signs, recovery.*

J. E. D., æt. 9 months. Was admitted Leeds Infirmary, Oct. 25, 1867. Well till four months ago. Since that time has begun to lose flesh, and to appear more heavy than natural. For one month has been subject to occasional vomiting, and seems to have pain in the head. These latter symptoms still continue.

O. S. There appears to be congestion, and slight infiltration of the right disk. Left disk as right, but less in degree.

R. pot. iod. gr. i., ter. die. Nov. 21, pt. pot. iod. and ol. morrh. ʒj. bis die. Under this treatment the child gradually improved, and on Dec. 19 was ordered syr. fer. iod. ʒ. x., with ol. morrh. ʒj. ter. die. With some occasional checks the improvement was on the whole maintained.

O. S. On Jan. 16 it is noted, 'the left disk whiter than right disk, which is red, and the vessels too distinct.'

The improvement steadily continued on the whole until March 12, 1868, when there was some return of the vomiting and occasional screaming, so that one day the mother feared a fit was coming on. The head is decidedly larger than it should be. There is no strabismus. The treatment was continued and the bad symptoms passed off; so that on April 19 a very good report was given. There had been no nausea, and no screaming or pain in the head. The disks had not changed. In a few weeks after the mother asked for a discharge, stating that the child was quite well. It has since been lost sight of.

24. *Hyperæmia of disks, suspected meningitis.*

Charles C——, æt. 11, admitted into the Leeds Dispensary on Oct. 12, 1868. Is a pale, weakly-looking child, with a tumid nose and lip. Has never been strong. Has only had one (sister) who is dead. Patient suffered from cervical abscess at the age of 9,

beginning in suppurating glands. Now complains of severe pain in the head of one month's duration. No vomiting. No fits.

O. S. The right optic disk is both pink and also a little filmy. Edges indistinct. The vessels are too full, but the arteries may yet be distinguished from the veins. Left disk as right, but more marked.

25. *Scrofulous eczema, headache, wakefulness, irritability, meningitis (?) , optic atrophy.*

Master G. S., æt. 11, was brought to Mr. Teale on account of his eyesight. His mother is 'delicate, and has a cough.' Patient several years ago had extensive scrofulous eczema on the head and behind the ears, and has always been weakly and liable to cough. About fourteen months ago became weaker than usual, his appetite fell, and he lost flesh seriously. He became also very wakeful. For about eleven months his sight has been noticed to fail, and during the same time, or longer, he has complained of violent headache, which 'throws him into rages.' He has also become fanciful and irritable, and at times he is a little delirious at nights. His mind cannot be called unsound, but he is 'unable to bear any schooling.' We found atrophy of both optic nerves, due no doubt to descending neuritis.

26. *Meningitis, atrophy of disks.*

Samuel W——, æt. 3, was admitted an out-patient at Leeds under the author's care in January, 1870. The family history is very unsatisfactory; the father is consumptive, and one child has died of 'brain fever.' Present patient never a thriving child, and of late has become very irritable. It wakes up suddenly and yells, and seems to have much headache. These attacks are often attended with vomiting. One month ago became subject to 'ditherings' in limbs, so that he falls down, or if in bed is generally convulsed. These attacks last two or three minutes, and may occur several times a day. This case might well have been called 'spurious hydrocephalus,' were it not for the

O. S. Examination. The child is so fretful, restless, and intolerant of light, that one disk (the left) only can be seen. It is distinctly atrophied, the retinal veins are dark and full, and there are remains of old exudations around the margins of the disk, signifying previous ischæmia papillæ or neuro-retinitis.

27. *Meningitis, recovery, amaurosis.*

H. K——, æt. 10 months, was admitted into the Leeds Infirmary, Sept. 30, 1870. Healthy until 'fever' and bronchitis six months ago. The 'bronchitis' broke down her constitution and left her very weak. Three months ago she began to have headache and vomiting. She also screamed out frequently with pain in the head, and became very irritable in temper. Latterly she had several attacks of convulsions. She then slowly recovered until a week ago, when the parents first became aware that the child was blind.

O. S. There is convergent strabismus of the right eye which was not congenital, and both disks show that state of atrophy with ragged edges and full retinal vessels which tells of past optic neuritis.

28. *Meningitis, retinal hyperæmia.*

Not to rely on my own cases more than necessary, I quote the following case of meningitis from some related by Dr. E. Long Fox, of Clifton; in this case only is there any record of ophthalmoscopic examination.

'Case XXVI. Mary R., æt. 23, single, acute headache, photophobia, tinnitus aurium. Vomiting at outset. Urine albuminous. No convulsion. Strabismus (slight) on 5th day, death from increasing coma on 7th day.

'O. S. Papillæ dull white on 3rd day with large vessels radiating from them. 5th day and 6th day, papillæ darker, and the vessels more numerous and larger than in health.

'Autopsy. Whole of arachnoid at base, and especially in fissures of Sylvius, thickened and granular, &c., &c.'

'St. Geo. Hosp. Reports,' iv. p. 82.

29. *Suspected tubercular meningitis, ophthalmoscopic signs negative, recovery.*

(Royal Hospital for Diseases of the Chest, City Road, under the care of DR. SANSOM.)

'Walter M., æt. 11, was admitted as an out-patient on November 16, 1867. He complained of shortness of breath, pain in the chest, and severe headache. Two years before his admission, whilst at school, he complained of dulness of sight. For twelve months

he has been losing flesh. He has suffered from headaches, which latterly have increased in severity, and they now give rise to crying and fretfulness, and the height of these paroxysms is described as "dreadful." The hereditary tendencies are as follow :—father suffers from chronic bronchitis ; mother has frequent headaches, so also have the brothers and sisters ; one sister suffers from rickets and general debility. The following describes his condition :—thin ; small flabby muscles ; pale ; head large and flattened ; teeth much notched ; high arch of palate ; slightly deficient resonance in left apex of chest ; here dry râles, and respiration harsher than in right. Ordered counter-irritation to the chest by turpentine liniment ; one grain of iodide of potassium and half-an-ounce of infusion of bark three times a day. No improvement followed ; on the contrary, the headache became frightfully intense ; it occurred every afternoon at two o'clock, and was accompanied by screaming.

'Considering the emaciation and the physical signs suspicious of an early stage of pulmonary tubercle, Dr. Sansom was led to fear that the violent headache might be due to incipient intra-cranial tubercle. To investigate this point he made an ophthalmoscopic examination. He found that the fundus of each eye was rather paler than usual ; the optic entrances were of their normal colour, and the vessels were small. In each eye the appearances were perfectly similar. This examination tended to negative the idea of tubercle in the meninges ; for it would be probable in such case to discover hyperæmia instead of anæmia, and the perfect similarity of each would not obtain.

'The following treatment was adopted :—cantharides blister behind each ear ; ten grains of bromide of potassium, afterwards increased to fifteen grains, three times a day ; cod-liver oil twice a day.

'The boy gradually improved, and on January 18 the note states, "He is mending very greatly, is livelier, and the pain in his head has greatly disappeared." The pulv. sodæ c. ferro of the hospital Pharmacopœia was added to the treatment.'

'Lancet,' Feb. 1, 1868.

30. *Tubercular meningitis, no change in the optic disks, mischief found to be confined to the posterior regions of the encephalon.*

——, a little child under the care of Mr. Carter of Stroud, suffered from all the symptoms of tubercular meningitis, followed

by death. Mr. Carter forwarded to the author the following notes of the autopsy.

‘*Stroud, March 20, 1868.*

‘After the death of my little patient the other day, I opened his head, and found the pia mater of the surface of the hemispheres intensely congested, the sinuses of the dura mater gorged with blood, the convolutions somewhat flattened, and the quantity of cerebro-spinal fluid increased. There was no increase of fluid in the ventricles, no trace of congestion of the optic nerves, and no mischief at all about the anterior part of the base of the brain. There was tubercular deposit and inflammatory adhesion, small in quantity and slight in degree, about the cerebellar pia mater, especially between its hemispheres. I could not open the orbits and follow down the sheaths of the optic nerves; but the autopsy showed that there could have been no change in the eyes beyond some impediment to the return of the blood; and of this I could see no sign, long after all doubt about the nature of the case had ceased.’

31. *Tuberculosis, meningitis, amaurosis, death.*

‘A smith, æt. 45, enjoyed good health up to æt. 32. Comes of a tuberculous stock; became bronchitic, lost his appetite, and at 40 had to leave his occupation. At length, in addition to extensive lung mischief, symptoms of tubercular meningitis set in. Head-ache and loss of memory, &c. had existed two years, and loss of vision began six months ago, and ended in blindness in four months and a half.

‘O. S. Exam. Irids contracted and immovable. Media clear. Both disks swollen, steep, muddy, and remarkably red. Neither scleral nor choroidal edge visible, the surrounding retina dull, the arteries thin and the veins abnormally wide, and thrown into strong curves.’ (*Ischæmia Papillæ. T. C. A.*)

Pagenstecher and Sämisch, ‘*Augenheilanstalt zu Wiesbaden*,’ Part i. 1861, pp. 53, 54.

I presume an autopsy was made, as the case is recorded as meningitis, but no details are given. Such a conclusion of pulmonary tuberculosis has often occurred in my practice. T. C. A.

32. *Meningitis diagnosed by the optic signs, autopsy.*

Madame X., æt. 28, admitted into the Hotel Dieu, March 31, 1866. Complained of violent supra-orbital pains, chiefly at night. No fever. Pulse slow and regular. No vomiting, indigestion or constipation. Intelligence perfect and no suspicion of meningitis arose. M. Peter failed to relieve her by antisyphilitic means. Galezowski examined the eyes.

O. S. Margin of disks concealed by infiltration; a small hæmorrhage at the upper and outer part of the right disk, some white streaks along the vessels. At a later period a hæmorrhage appeared in the retina of same eye and more white exudations. Diagnosis, meningitis of the base.

The patient's state had become aggravated, and death occurred on April the 28th.

Autopsy. No dropsy of the ventricles. Meningitis at the base of the encephalon. Many granulations. Miliary granulations found also in the lungs. Microscopic examination of the optic nerves. Their fibres were unchanged, but the sheaths were infiltrated, softened, and contained pus cells.

Galezowski, 'Etude Ophth. sur les maladies cerebrales,' pp. 136, 137.

33. *Meningitis, retinal hyperæmia, death.*

A. B., æt. 25, under Dr. Reynolds in University College Hospital. Admitted November 10, 1869. Had headache, fever, restlessness, twitchings of face and upper limbs. Died comatose on the third day after admission.

O. S. On admission pupils slightly dilated and acting well to light. Hyperæmia of both retinæ, more marked in the left.

Autopsy by Dr. Bastian.—'Surface of arachnoid dry and sticky. Convolutions of vertex notably flattened. Some small yellowish white patches of lymph here and there along the vessels; very slight, however, on vertex, but more marked on vessels emerging from each Sylvian fissure. Base of brain lined by a thick layer of greenish yellow lymph, from optic commissure back over pons and whole of medulla oblongata, laterally to adjacent surface of cerebellum. No obvious tubercular granulations on vessels. Ventricles distended notably, and corpus callosum diffuent. White substance of hemispheres softer than natural.'

'Medical Times and Gazette,' June 18, 1870.

34. *Tubercular meningitis, vomiting, ischæmia of disks, dropsy of nerve sheaths.*

A lad, æt. 16, was under Manz with headache, vomiting, &c. He found the disks swollen and hyperæmic. The patient was unconscious, and died in four days. The pia mater at the base, especially about the chiasma, was infiltrated and covered with layers of fibrin. There was miliary tuberculosis here and elsewhere. The optic nerves were from twice to thrice their natural size, owing to distension of their sheaths with serous exudation. The nerves themselves were normal up to the sclerotic. The disks were swollen and muddy.

The retina and choroid were normal.

Manz, 'Zehend. Klin. Monatsbl.' iii. 1865.

35. *Meningitis (?), optic atrophy.*

No. 12. Charlotte T., seen Oct. 1, 1866, æt. 7. Fair complexion, red hair, large head, thick alæ nasi. Could walk well, was cheerful but nervous. Illness began Jan. 1, 1866. Pains in limbs, sickness, convulsions, loss of speech for an hour or two at a time when recovering from the convulsions, delirium at times, complete paralysis of legs, and for a long while inability to bend her back, stiffness and pain in neck. In five weeks began to get about again, and it was then noticed that the eyes were crossed. *She was supposed to see well.* Her hair all came off. In a week or ten days the left eye was found to be failing. In a month the right eye failed also.

Pupils large, sluggish.

O.S. White disks (dirty white) abruptly margined. No disease at yellow spot. The large veins and arteries somewhat diminished in size.

Hutchinson, 'Royal Lond. Oph. Hosp. Reports,' vol. v. Part iv. p. 313.

36. *Meningitis (?), optic neuritis.*

'No. 8. Wm. Fisher, seen May 28, 1866, æt. 13. He could walk quite well. History of fits and "dreadful pain in his head" in February. Was admitted into St. Thomas' Hospital (Dr. Bristowe) in March. At this time mother said the right eye looked larger than the other. In the hospital he got much worse, and was expected to die. When discharged he could not walk; he did not

seem to have any use in his legs. Both pupils were motionless, the right being twice the size of the left.

‘O. S. In both the optic disks were dirty and fluffy, the margins being concealed ; the arteria centralis much diminished in size, and the vessels on the disk being concealed in part by the deposit of lymph. In both the appearances of neuritis were passing off, but the right was further advanced in atrophy than the left.’

Hutchinson, loc. cit.

37. *Meningitis, optic signs.*

The following case was placed in the author's hands by his friend Mr. Seaton, of Leeds :—

‘ — Butler, æt. $3\frac{1}{2}$, delicate, had tender eyes, and could not walk till æt. 2. Dec. 14, 1870, was seized with a fit, with subsequent drowsiness. 15th. Still drowsy and apathetic, but conscious when spoken to. 16th. Same state. No heat of head. 17th. More unconsciousness. Pupils dilated and inactive.

‘O. S. Brilliant choroidal glow ; optic disks in both eyes very red, so as not to be readily distinguishable. Retinal vessels normal in course and calibre, but they had lost their defined outline, and had a “furry” appearance. (Edema ? T. C. A.) Near left disk a cloudy patch on the retina.

‘During the next two days the head became hot, the eyes injected, and the child died with strabismus and coma.’

38. *Traumatic meningitis in a horse, optic atrophy.*

Mr. Fearnly, a very intelligent veterinary surgeon, in Leeds, has made frequent use of the ophthalmoscope in treating animals. A horse was brought to him, said to be suffering from lameness, but he was able to assure himself that the animal was staggering from disease of the encephalon. He examined the eyes, and was surprised to find the nerve entrance quite white, and all the vessels ‘as fine as wires.’ The animal he then found had had three epileptic fits. These were repeated, and death occurred in a few days. On opening the head he found injury to the occipital bone, and traumatic meningitis all along the base, the membranes seeming to be ulcerated in many places.

Communicated by Mr. Fearnly to the author.

39. *Tuberculosis, rheumatism, meningitis (?), convulsions, retinitis.*

C. W., æt. 26, complains of failing sight. Both in herself and in her family there is a marked history of tuberculosis. Two years ago she had rheumatic fever, and had at that time much pain in the head, which has continued ever since. Seven months ago had a fit, slightly affecting the right side ; has had many since, often six or seven in a week. No heart mischief. Dulness at apices of both lu. gs.

O. S. Feb. 4, 1868 : Both disks were found slightly raised, and very pink, scarcely to be distinguished from the retinas. Reads 10 Jäg. with right eye, 16 Jäg. with left eye. March 2 : Position of right disk known only by convergence of vessels. Retina very hyperæmic, and silvery films are forming upon it. Left eye as before.

This girl was probably suffering from meningitis, tubercular or rheumatic. I have notes of two other cases, in which there was reason to suppose that meningitis had complicated rheumatism, and in which there were remains of optic neuritis.

C. W. ceased to attend upon the author and Mr. Oglesby, and has been lost sight of.

40. *Eruptive fever, meningitis, idiocy, old mischief in and about the optic disks.*

M. J. A., æt. 4. Was quite well and intelligent up to fifteen months ago, when she had an eruptive fever (scarletina?). Before convalescence was secure she was seized with headache, vomiting, and convulsions. After many weeks these symptoms slowly subsided, but have not yet disappeared. At this time her mind was found to be much impaired also.

O. S. The disks are dirty white and blotchy. Traces of old effusions about their margins, and along the course of some of the vessels. A few veins in both eyes are very dark and tortuous. Sight is thought 'not to be very good.' Under iodide of iron and cod oil the child improved in general health, but still vomiting and slight convulsions reappeared from time to time. Her mind, however, deteriorated still more. She quite 'lost her head,' and became very mischievous. She remained under the author's care about six months, the only change in the disks being that the large veins slowly diminished in size. The vomiting and fits seemed wholly

to disappear, but in all probability the child would find her way into an asylum.

41. *Scarlet fever, meningitis, sudden death.*

In January, 1870, the author was asked by Mr. Hopkins to see a little child, æt. 4 years, who had been attacked, three weeks before, with scarlet fever. His recovery had been fair and without albuminuria, but there was some swelling tending to suppuration at each angle of the jaw, and during the last week head symptoms had crept on. I found a 'strumous'-looking child with large head, almost immovable pupils, and fretful, hyperæsthetic manner. I learned that he had become drowsy and wildly delirious at night, that he had intense pain and heat in his head, nausea and occasional vomiting, and once or twice transient strabismus. The sub-maxillary swellings were opened by Mr. Hopkins and some grumous matter let out. There was no serious mischief in these parts. The continued use of mild purgatives had acted very beneficially upon the head symptoms.

O. S. Veins of both retinas very full and dark, and slight effusion obscuring the disks.

I hoped this might only signify encephalic congestion, and this hope seemed to be strengthened by the apparent success of the purgations. We ordered quinine and iodide of potassium. The child seemed to rally, again relapsed, rallied again, but finally had another severe relapse, in which he died, after the manner of that deceitful disease, meningitis.

42. *Scarlet fever, meningitis, hypercæmia of one disk.*

Rebecca R——, æt. 10, was admitted under the author in August, 1867, and was a long time under notice. A few months before, when in excellent health, she had scarlet fever rather severely, and had a discharge from the right ear. On admission, this ear, as tested by watch, was deaf, the watch could scarcely be heard at one and a half inch, which in a few weeks she was able to hear at eight inches. There were many symptoms of local meningitis at the base, such as headache, sickness, strabismus (external) of right eye, &c., which I need not detail at length.

O. S. Right disk very pink, vessels full, edges indistinct. Left disk normal, or nearly so.

Reads No. 6 (Jäger) only with right eye ; reads No. 2 with left.

This child remained a long time under my care, and under the use of iodide of iron and cod oil, with the occasional use of chalk and mercury, she completely recovered. Her mental faculties, previously much weakened, were restored; her right disk cleared, and the right ear became sensitive to sound. She was threatened, however, with pulmonary symptoms when she was discharged on the family removing elsewhere.

43. *Scarlet fever, otorrhœa, meningitis.*

G. H., æt. 11. Scarlet fever at æt. 2, followed by discharge from ears, which still (July 2, 1867) continues. Eight years ago had 'brain fever,' said by the doctor to be due to the otorrhœa. He is decidedly deaf. At present suffers from symptoms of meningitis; is at times strange and almost maniacal in manner; has attacks of intense headache; nausea; occasional vomiting; convulsive attacks; intolerance of light; transient debility of limbs; inability for mental work. These symptoms vary a good deal in frequency and intensity; sometimes is free for several weeks, when he will suddenly run into the house with a violent access of headache. Is always best when ears are discharging freely; they discharge most freely when the boy is warm. The whole neighbourhood of the left ear is puffy and tender, especially behind the pinna. In both disks is well-marked ischæmia. The bright œdematous disks stand out steeply, and the vessels ride over them, and the veins are full and dark.

Sept. 17. Has been treated with iodide of potassium, cod liver oil, and small doses of corrosive sublimate. Is better. Has had one fit with unconsciousness. Has still morning nausea. The disks have receded, leaving a more plain surface; their edges are undistinguishable; the vessels are large and dark, and there is still some œdema.

Oct. 15. Much as before. Disks flatter and less œdematous.

Dec. 24. On the whole much better. Disks as last report. Yesterday, however, had intense pain in head; screamed and rolled on the floor. Has lately been taking syrup of iodide of iron and cod liver oil.

Dec. 31, 1869. Has recovered a good deal from his head symptoms, and his ears are much less troublesome. There is now but little discharge. The edges of the right disk have cleared all round, except quite at the inner edge. The left disk is indistin-

guishable, but the vessels are not distended. Sight in both eyes good, and always has been. Has a cough, and there are evidences of degeneration at both apices.

44. *Erysipelas, delirium, amaurosis.*

'On Sept. 18, 1862, a girl named Cultur, æt. 14, came to the Clinique of M. Desmarres. Her sight had been weak for three months. Three months ago she suffered from erysipelas of the head, with violent delirium; and on recovery her vision was found to be defective, and she suffered from constant pains in the frontal region.

'O. S. Atrophy of both disks was found.'

Bouchut, loc. cit. p. 246.

45. *Optic neuritis, or ischæmia papillæ, in pyæmia.*

(Letter from DR. CLIFFORD ALBUTT to the Editor of the
'Medical Times and Gazette'.)

'SIR,—In your journal of last week, Dr. Hughlings Jackson writes to ask whether other ophthalmoscopic observers agree with him in reporting changes of the optic disk in pyæmia. He appears from his expression "swollen disks" to have noticed the condition I have ventured to call ischæmia papillæ. I entirely agree with him as to the occurrence of ischæmia papillæ or of optic neuritis in pyæmia, as in other diseases of like character where meningitis may occur. I have little hesitation in saying that meningitis was present in the cases Dr. Jackson records. He himself observes that the state of the disks was like that which he had observed in a case of tubercular meningitis, and in a case of syphilitic disease of the base, which again was probably more or less meningitic in character.

'In support of the co-existence of optic neuritis or ischæmia papillæ with pyæmia, and in support of the proposition that meningitis is the immediate cause of these states, I will bring forward a case which came recently under my notice, and for which I am indebted to Dr. Crichton Browne, of the Wakefield Asylum, as I am indebted to his great and untiring courtesy for very much more of my pathological and other experience. A patient recently died in the Wakefield Asylum from pyæmia, and Dr. Browne, who was present at the autopsy, was kind enough to note particularly for

me the state of the optic nerves. Scattered pyæmic abscesses were found in some numbers in both lungs, a number of minute abscesses in the kidneys, blood-stains upon the heart and valves, and other clear evidences of pyæmic blood-poisoning. There had been much delirium before death, and very marked meningitis was found about the base of the brain. The optic nerves also were found to be very vascular and diminished in consistency, the chiasma and branches were affected up to the eye, and backwards the same changes were found in the tracts and up to the corpora quadrigemina.'—'Medical Times and Gazette,' June 27, 1868.

46. *Enteric fever, meningitis, paralysis of cranial nerves.*

James O'B., æt. 7, admitted under the author's care into the Leeds Infirmary on April 15, 1870. Had enteric fever very severely the October preceding. Had severe headache, vomiting, and intolerance of light, with slight convulsions during the fourth week. On convalescence, was noticed to have external strabismus of the right eye.

O. S. Now some deficiency of vision, and commencing atrophy of both optic disks; ptosis of both eyelids, in right eye almost complete; and external strabismus of right eye.

N.B. I have five cases of optic atrophy following continued fever with severe head symptoms, but they do not present any important differences from the foregoing. T. C. A.

47. *Hydrocephalus, ischæmia of disks, and subsequent slight atrophy.*

W—— M——, æt. 15 months, admitted out-patient under the author on January 17, 1868. Always delicate, but 'ailed nothing' till two months ago, when he began to be fretful and sometimes complained of headache. There was no night screaming, vomiting, or strabismus. The head has been slowly enlarging since, and is now decidedly but not greatly swollen. The mother is delicate and has a cough.

O. S. Examination. Both disks prominent and vascular, but tolerably transparent. Edges invisible, but no exudation extending upon the retina or concealing much of the vessels. (Vide 'Description of Ischæmia papillæ.')

(Stauungs papilla, noted on card). Under long and careful

treatment with cod oil and the iodides, the child improved very much, and the head lessened by two inches in circumference.

O. S. Examination, March 12. The disks have gradually subsided, but are whitening. The sight seems certainly very defective.

O. S. Examination, July 11. Disks greyish white, edges blurred. Sight much improved. Discharged 'cured.'

48. *Hydrocephalus, injured disks.*

H. T., æt. 8, admitted under the author into the Leeds Infirmary Jan. 12, 1868. His head has been slowly enlarging for five or six years. At present decided hydrocephalus, but not extreme.

O. S. Optic disks smudgy, as if there had been extensive effusion in and near them some time ago. The evidence of interference is very decided, and the disks look grey, but the child is not blind. The father however, when questioned, says the child's sight is certainly somewhat defective.

49. *Hydrocephalus, destruction of disks and retinas.*

A. G——, æt. 5 months, admitted into the Leeds Dispensary on March 31, 1868. Has one brother, aged $2\frac{1}{2}$ years, who is quite healthy. None dead. Mother has a cough and 'is delicate.' Present child born healthy. At two months old it 'began to roll its eyes about,' and the head began to enlarge. There has been no great disturbance of general health. The vision is supposed to be very defective or nearly absent.

O. S. Both retinas show well-marked patches of exudation, some brown and dotted with pigment, others white. Disks both quite white. The vessels do not seem much changed, but the examination is difficult.

50. *Hydrocephalus, atrophy of disk.*

James K——, æt. 1 year, admitted into the Leeds Dispensary on July 27, 1868. Two or three months ago was noticed to be much ailing, to be restless and sleepless, and the head began evidently to enlarge. This has been especially noticed during the last month. It is now obviously of too great bulk. The child is very irritable, but there is no marked vomiting. The brother died of 'water in the head and lung affection,' and 'this is getting like him.' Both children seemed healthy when born. There are older children living, reported healthy.

O. S. Left disk only seen, as the child was very 'fractious;' this disk was white and blurred at the edges. The retinal vessels were large, but not very greatly distended.

51. *Hydrocephalus, atrophy of disks.*

B. S. —, æt. 11 months. Admitted into the Leeds Dispensary on Sept. 7, 1868. The head has been noticed to be decidedly growing too large for six or eight weeks. Ten days ago occasional vomiting set in, occurring about once a day, and 'purposeless.' The legs seem weaker than natural.

O. S. Both disks are whiter than natural, probably atrophic. The retinal vessels are full, and seemed to point to a past stage of congestion.

This patient did not attend again.

52. *Hydrocephalus, commencing change in disks and retinal vessels.*

——— ———, æt. 1 year and 9 months, was brought to the author on Oct. 26, 1868, with symptoms of hydrocephalus. The disease had come on somewhat rapidly, the child's head having become evidently large within three or four weeks. Occasional vomiting for one month. Nutrition somewhat impaired. The head is not greatly increased in size, but is decidedly larger than normal, and the forehead is a little bulged.

O. S. In both eyes there is some vascular change. The retinal vessels are a little too large and some of them are tortuous. The disks are very pink, and the small vessels nearing the disks rise a little over an elevation on one side, showing a slight degree of ischæmia papillæ in both.

53. *Blow on head, concussion, left hemiplegia, paralysis of orbital muscles, optic neuritis, death, meningitis.*

A little boy, supposed to have fallen down stairs. When seen by Mr. Hutchinson was wholly insensible; left limbs weak; of right eye the pupil is dilated, the ocular muscles all paralysed, the conjunctiva congested, and there is neuritis of the disk. The head had been injured three weeks before marked symptoms of cerebral disease set in, though during all that interval he had been heavy and stupid. Then came on shivering, sickness, and constipation. He

died a few days after Mr. Hutchinson saw him. The family attendant, Mr. Owen of Leatherhead, made the autopsy, and found that there was, as had been diagnosed, meningitis at the base on the right side.

Hutchinson, 'R. L. O. H. Reports,' vol. v. p. 108.

54. *Blow on head with cricket-ball, subsequent head symptoms, meningitis (?), death.*

On the 30th of August, 1869, the author was requested by Dr. Burnie of Bradford to see Master C., who was suffering from obscure head symptoms. I found a case which much resembled those which often follow railway and like accidents. The boy, who was naturally healthy and strong, had been struck on the head, when at school, by a cricket-ball. This occurred during the spring half-year, about the beginning of May. He was felled to the ground and remained there, stunned; he was lifted up and taken into the house, when he seemed to recover as from an ordinary concussion. He seemed well for five or six weeks, when he began to droop. He was irritable, unable to apply himself to work, sleepless and moody. These symptoms got worse, and he was brought home. In addition to the above symptoms, he now complained of headache in the occipital region (where he was struck), and of nausea with occasional vomiting. When I saw him, his general health, strength, and nutrition had failed so much that he was almost confined to bed, and could scarcely read the lightest books. He had never had any strabismus or convulsion, but had once or twice suffered from startings in the body and limbs.

O. S. The pupils did not act well, but were equal. The veins of both retinas were very full, and a good deal of serous effusion in the inner half of both disks. Both disks were veiled with a film of serosity.

I explained to the father that ophthalmoscopic signs were yet too little understood to be of certain value, but that I believed there was serious head mischief. This the event proved, for the boy died some weeks later with symptoms of compression. No post-mortem could be obtained.

55. *Two cases of concussion with amaurosis.*

'(1) P. A., a locksmith, æt. 32, fell from a ladder upon his head six weeks ago. Was unconscious for twenty-four hours. Amau-

rosis of the left eye then set in, with headache, but no other palsy appeared. It would seem that the mischief lay between the eye and the chiasma.

‘(2) Miss E., æt. 18, fell down three steps three weeks ago, and was taken up unconscious, and remained for twenty-four hours. Then came on severe headache and spine-ache with paralytic symptoms on the right side. The sight and mental faculties (memory, &c.) also declined. On admission seems stupid, and takes no notice. Palsy of movement on right side.

‘O. S. Both disks turning white.

‘No farther account is given.’

Pagenstecher, ‘Augenheilanstalt zu Wiesbaden,’ Part iii. 1866, pp. 77, 78.

56. *Concussion, amaurosis, meningitis?*

‘A peasant, æt. 55, fell fifteen feet on to a barn floor; he was brought home and put to bed unconscious. This was six years ago. He recovered slowly and incompletely; the head had suffered chiefly, and a depression remained. He had feverish attacks, with vomiting, dulness, and incapacity for work. His sight also failed, chiefly on the right side. When seen there was white atrophy of the right eye, and loss of the inner and upper quadrant of the field of vision. The left eye seemed normal. There was palsy also of the right rectus internus. At the angle formed by the sagittal with the lambdoid suture on the right side of the skull was a depression the size of a gold piece, and one and a half lines in depth.’

The patient seems to have obtained some relief from treatment, and to have returned home, which suggests that the internal mischief was rather chronic meningitis than sclerosis.’ (T. C. A.)

Pagenstecher and Sämisch, ‘Augenheilanstalt zu Wiesbaden,’ Part i. p. 67.

57. *Blow on head, tumour of right posterior lobe, amblyopia.*

In the last year of the American war a man, æt. 23, was struck upon the head with a splinter of a shell, which stunned him.

Mental defects, restlessness, severe attacks of headache, vomiting, and latterly strabismus, were the chief symptoms which followed. In addition to these the pupils were dilated, and there was amblyopia. After death a cheesy mass was found in the upper part of

the right posterior lobe, which was glued to the membranes and to the calvaria. The seat of this mass corresponded to the place of the blow.

Lomax, 'Philad. Med. and Surg. Reporter,' July 31, 1869.

58. *Blow on head, amaurosis, autopsy, sclerosis of brain.*

'A young lady, æt. 15, received a tap rather than a blow on the right side of the head. It gave her at the moment rather severe pain, and for thirty years she continued subject to headaches, commencing in the part struck. She then, though naturally very lively, began to grow heavy, and sometimes stupid and sleepy. For the last year and a half of her life it was difficult to keep her awake, but when she was awake, though it was but for half an hour, she displayed all her natural brilliancy of conversation. She became completely comatose, and died convulsed. Her vision had become very much, though very gradually, impaired.

'*Autopsy.* Bone, at part struck, of very dark colour over a space equal to a crown piece; that part of the parietal bone being in fact transparent, and almost absorbed. Its dark colour was derived from the hemisphere under it, which was black, and the dura mater was absorbed. The portion of brain under the injury was indurated and scirrhus, and this change had taken place through the whole of the middle lobe of the cerebrum. The optic nerves were compressed and as flat as tape.'

Quoted by Mackenzie, from Howship's 'Surgery.'

59. *Blow on head, amaurosis, autopsy, sclerosis of brain.*

'A young gentleman, æt. 12, was struck on the right side of the head, and an obstinate wound resulted, and remained for six years. It then healed, and soon he perceived that his sight was failing. He then became epileptic and blind. He was trephined, without discovering disease of scalp, skull, or membranes; the pupil now became sensitive, apparently owing to the escape of some blood and serum, but blindness remained. Fever now came on, and he died soon after.

'*Autopsy.* Cranium healthy; so was the dura mater. Below the place of the operation the pia mater was found to have evidently suffered from chronic inflammation over a circumscribed space. On cutting into the brain it was found indurated to a considerable

degree, and this induration had extended itself to the whole of the middle lobe of the cerebrum, commencing upon the surface of the hemisphere, and passing through the brain down to the basis of the cranium.'—Quoted by Mackenzie, from Howship's 'Surgery.'

60. *Blow on head, aphasia, autopsy, sclerosis of brain.*

John W——, of Drighlington, was struck down in a fight, and fell with the left temple upon an iron plate. He was stunned and sick, but was able to walk home (about half a mile). From the moment of the fall he was dumb, and never for the rest of his life could say the commonest sentence. He was otherwise bright and intelligent, and pursued his calling as a cap-hawker. He was under the author's observation for some time, but nothing wrong was to be found save the scar on the temple and the aphasia. He could only say three or four words, and these he used quite irrelevantly. About nine months after the accident his sight failed, and the disks atrophied, but he never became blind. He died about two years after the fall, having become epileptic about nine months before death, and hemiplegic on the right side about three months before death. Dr. Sykes of Drighlington was so kind as to make great efforts to get an autopsy, and he succeeded. We found no disease of bone or parietal dura mater. Over the upper part of the left anterior lobe there were marks of old meningitis with thickening and adhesion, and the whole of the lobe, including Broca's convolution, were sclerosed, and presented the microscopical characters of that change. The left striate body was much softened. The optic nerves were decidedly atrophied, but not compressed.

61. *Fracture of base of skull, retinal changes.*

'The patient was a man who had a blow upon the head, by which the base of the skull was fractured. Jacobi examined the eyes several times during life, and also after death. He found on the eleventh day after the injury large yellowish white patches with small extravasations of blood, and after death he noted conglomerations of granule masses in the middle layers of the same parts of the retina.'—Jacobi, A. f. O. xiv-i. 147-149.

N.B. I presume that Jacobi could not have overlooked albuminuria had this been present. T. C. A.

62. *Fracture of base, amaurosis.*

‘M. G., æt. 23, fell heavily, face downwards, upon the pavement. Unconscious for two days, although he was promptly bled. Left eye red with considerable sub-conjunctival hæmorrhage; the apophysis ascending from the left upper jaw presents a dislocation upwards, and projects near the punctum lachrymale. The bones of the nose are broken. Left eye blind and pupil dilated; and at first the eye was prominent and the sixth nerve palsied. The cheek is insensible. The disk is atrophied. In one month health was restored, and the right eye remained good, but the left remained blind. Evidently there was a fracture near the foramen which gives passage to the optic nerve, and which was thereby compressed and atrophied.’—Galezowski, *loc. cit.*, pp. 142, 3.

63. *Fracture of base of skull, ecchymosis into retina.*

‘E. C., carpenter, æt. 54, fell upon his head two days before admission. Unconsciousness one hour, dizziness, and vomiting the rest of the day. There is much ecchymosis about the right fronto-parietal suture, at the root of the nose, in the eyelids, and conjunctivæ. Right eye absolutely blind.

‘O. S. Its fundus is so red that the vessels are hardly to be seen: at the inner and upper part of the disk is a striped hæmorrhage; it is oblong, and directed upwards and inwards, being to the disk as a tangent to a circle. Above and below it are two little whitish streaks due to slight infiltration around the morbid product. Disk intensely red, the vessels dilated; one of them ends in the hæmorrhage which probably plugs its ruptured end.

‘There was a large escape of cerebrospinal fluid. The man recovered, but the eye remained weak.’

Bousseau, ‘*Des Retinites secondaires*,’ p. 97. Paris, 1868.

64. *Fracture of the skull, depressed, and probably compound; recovery, followed by optic neuritis and atrophy.*

‘Under the care of Dr. Slack, we saw a girl, æt. 22, who had become blind after a fall on the head. She stated that, five months before our visit, she fell and struck her head against some sharp portion of a wheel. She was insensible for about two hours, and her nose bled. A day or two afterwards her eyes became blood-shot. She was troubled with sickness for several days after the

accident, but did not seem to have vomited blood. There was neither deafness nor discharge from the ears. About four weeks after the accident she suffered for several days from headache and sickness; and about the same time her sight began to fail, the right eye being affected first. She stated that, simultaneously with the failure of the right eye, she partially lost power on the right side; but that she afterwards quite regained it. The left eye afterwards became affected in the same way as the right. She had no medical advice for the injury to her head, but stated that there was a wound of the scalp which bled somewhat. When we saw her there was a depression, admitting the tip of the index finger, in the scalp, nearly over the supero-posterior part of the right parietal bone. She was quite blind. The pupils were widely dilated—the right somewhat more so than the left. In the right eye, the retinal veins were congested and somewhat tortuous; the disk was pale, and its margins very indistinct, from the existence of semi-opaque effusion, which, however, did not completely hide the vessels at any point. In the left eye there were a crescent and myopic refraction; the retinal veins were markedly less distended than in the right eye; the disk was quite white; and there was no trace of effusion.

‘We are indebted to Dr. Haynes, the house-surgeon, for the opportunity of seeing this case.’

‘British Medical Journal,’ March 12, 1870.

65. *Abscess of right hemisphere, amaurosis, autopsy.*

‘A boy, æt. 4, under Dr. Peacock, had been ill three months with convulsions and subsequent left hemiplegia. He had also ptosis of right eyelid and dilatation of pupils; the right eye was blind, and the left eye became enfeebled. He died in convulsions about three months later, the ptosis having disappeared. An abscess containing eight ounces of pus was found in the right middle cerebral lobe extending close to the convolutions. This was surrounded by indurated tissue [the depth of which is not mentioned] containing compound granular corpuscles, and a few similar corpuscles diverted in the right corpus striatum and thalamus. There was some sub-arachnoid effusion and lymph on the surface of the right hemisphere. Left side of brain, spine, and other organs healthy. Dr. Peacock remarked upon the perfect preservation of intelligence.’—‘Pathol. Transact.’ vol. xvii.

66. *Abscess of posterior lobe, hydrocephalus, amaurosis.*

'A young girl, æt. 5 years, was seized with general convulsions eight days after the disappearance of measles. She had headache on the right side, mouth drawn to the left, strabismus, and left hemiplegia. During a few days at first she had thirst and heat of skin. The palsied limbs are contracted and stiff, the others constantly in movement. There was continual irritability, no stupor or coma, hearing good, sight gone, pupils dilated; the strabismus was of the right side, and convergent. Latterly there was insensibility and stertor. Death in three months.

'Autopsy. Dura and pia mater adherent on the right side. Right hemisphere swollen behind, the cerebral substance here being firm and leathery. The posterior lobe was almost filled by a globular sac which can be easily separated from the cerebral substance. It contained four ounces of laudable pus. The ventricles were distended with fluid.'

Bateman, 'Edinburgh Med. and Surg. Journal,' 1805, vol. i. p. 150.

67. *Abscess in right hemisphere, neuro-retinitis.*

'A girl, æt. 12, was admitted into Oppolzer's Ward in the winter of 1864. She had received a blow upon the head which had produced unconsciousness. Symptoms of cerebritis gradually came on, slowly increasing hemiplegia of the left side, with convulsions at first of the palsied side, and afterwards of both sides. Facial paralyses were noticed, sometimes on one side and sometimes on the other; the left pupil was dilated, and the superior and inferior recti muscles were weakened. Amaurosis set in suddenly, and neuro-retinitis was discovered. An abscess was found at the autopsy, which was seated closely over the right optic thalamus.'

Benedikt, 'Electro-therapie,' p. 257.

68. *Softening of brain, amaurosis.*

Rostan ('Recherches sur le Ramollissement du Cerveau,' 2nd ed. obs. ii. pp. 28-31) records a case of softening of the brain. 'The optic nerves were flattened, diminished in calibre, in a state of atrophy, of reddish appearance, like a small arterial tube, and without any resemblance to the whitish cord which they ordinarily present.'

Quoted by Lawrence, p. 501, who does not say more about the softening.

69. *Hemiplegia, convulsions, neuro-retinitis, death, mesocephalic softening.*

'A soldier, æt. 26, suffers from constant paroxysmal headache on right frontal region. Is feeble and moves slowly. Mind clear, save occasional hallucinations. Recent amblyopia.

'O. S. The retina near each disk had lost its transparency, and was grey and opaque, this opacity extending along the principal vessels, and at times obscuring them. Numerous retinal ecchymoses were scattered about the disks, growing less frequent near the macula lutea. In the course of the next six months left hemiplegia and convulsions were followed by death. A month before death there was no perception of light, but shortly before death light was again perceived. There are no notes of subsequent examinations with the mirror.

'Autopsy. Softening and reddish grey discoloration first observed over small space in crus cerebri, just anterior to pons varolii. This the finger readily followed, without apparent tearing of brain tissue, anteriorly into the white substance of the right hemisphere to the distance of two or three inches. On this side the optic thalamus and corpus striatum were so much involved in the morbid process as to be scarcely traceable. The softest portions were pulpy but not fluid; and irregular greyish, reddish, or dull white limited portions being also gelatinous and somewhat translucent. Under the microscope the principal and quite uniform appearances were broken down, curved, and distorted nerve-fibres, together with abundance of free fat granules distinct and agglomerated, rarely enclosed in cell walls. No pus or pyoid cells seen.'

Drs. H. Darby and Upham, 'Boston Med. and Surg. Jour.' v. 72, p. 21, quoted in 'Ophthalmic Review,' vol. ii. p. 78.

70. *Disease of cerebellum, amaurosis.*

Boy, æt. between 14 and 15 years; very tall of his age. Until six months before death had been quite healthy, living in south of England. No hereditary taint of any sort. Came to Manchester six months ago to enter business. A very quick, intelligent lad, very anxious to get on; learning French, &c.; growing very fast.

Six months ago began to have occasional bilious vomiting, recurring every three or four days. Eyesight soon began to fail, and gradually, in course of three months, became totally blind of both eyes. Flesh and strength gradually failed; but no actual paralysis

took place to the end of life. Trance-like attacks occurred once or twice a week, lasting from fifteen to forty minutes, with total insensibility, but without any convulsion. No irregularity of movement, staggering (or titubation), occurred at any time, nor any priapism. The trance-like attacks always followed the bilious vomiting; and all day after one of these attacks the lad would be heavy, dull, and indifferent, not originating any conversation, and without appetite or power to eat. On the days between these 'bad' days the appetite was excellent, or rather, voracious, and the intelligence remarkably brisk. He was cheerful, lively, laughing, wanting to be read and talked to, learning French, and altogether remarkably quick and smart (this applies to the last three months of life). Hearing very quick, but the hearing of left ear somewhat impaired. The pulse quick but regular. *Tongue* in last three weeks (being the time he was under my observation) clean and *fiery red*, *inclined* to be dry, but never actually dry, so far as I know. Patient lay at this time with mouth invariably and continually open (gobemouche fashion); perhaps this had something to do with the remarkable state of tongue. He kept his bed for last three months of life from exceeding weakness and *great* emaciation; nevertheless, he was taken home from infirmary ($2\frac{1}{2}$ miles) in a cab, sitting up. He could sit up in bed to near the time of death. Skin dry. Urine contained neither sugar nor albumen. No chest symptoms. Occasional violent headaches occurred, but nothing remarkable or constant. He became insensible in last six hours of life.

Died Nov. 25, 1866. *Autopsy*. All organs outside skull healthy (lungs, heart, liver, &c.) *Cerebrum* also healthy, but cerebellum extensively diseased; both lobes softened—spotted on the surface with small white spots—the left lobe *less* diseased than the right. The right lobe gave way on removing; its central parts were very soft, the white matter in places of consistence of cream. Under microscope the nerve fibres are found totally destroyed in the softened parts, and reduced to granular matter, with *immense* quantities of 'granular corpuscles,' and also numerous free oil globules. The presence of 'granular corpuscles' could be traced into the crura cerebelli, mixed with healthy-looking nerve fibres.

The white spots on surface were found to be masses of 'granular corpuscles.'

There was general fatty degeneration of arteries at base of brain.

Communicated to the author by Dr. Roberts of Manchester.

71. *Hæmorrhage pressing upon the chiasma, blindness, autopsy.*

‘Mr. Stevenson was called to attend a patient between forty and fifty years old, who was found lying on the ground in an apoplectic fit. The pupils were dilated, and he was quite blind, but not destitute of sensation and feeling. He expired within twelve hours after the attack.

‘*Autopsy.* Decisive marks of meningeal congestion : large accumulation of discoloured serum in the ventricles, and a mass of coagulated blood so placed as to compress the optic nerves at the part where they decussate.’

Mackenzie, ‘Diseases of the Eyes,’ third ed. 1840, p. 874.

72. *Headache, vomiting, amaurosis, hæmorrhage into various parts of encephalon.*

‘A man, æt. 32, was seized at 10 a.m., while talking, with giddiness, headache, and vomiting. He died in eleven weeks. Amblyopia noticed in three weeks; eyes examined in tenth week, when latter stages of neuritis were found. No palsy, some slight mental confusion, which passed into stupidity two weeks before death. Autopsy : Large hæmorrhage into middle cerebral lobe, and a few specks in corpora quadrigemina.

Hughlings Jackson, ‘R. O. Hosp. Reports,’ iv. 248.

Dr. Jackson regrets that further details are not in his possession.

73. *Old mesocephalic hæmorrhage, amaurosis, autopsy, cyst of thalamus and corpus striatum.*

‘A woman named B— had suffered eight years before admission from right cerebral hæmorrhage, with left hemiplegia which passed off.

‘Twenty-five days before admission lost anew the use of the left limbs.

‘On admission was found to be incompletely amaurotic. In a few days she died, and the right striate body and thalamus were found to be occupied by a kind of cyst which was empty and flattened, and surrounded by softened nervous matter. The crus cerebri is wasted, and also the lower stage of the pons, the anterior column of the cord, and also the optic tract.’—Lancereaux, loc. cit.

Also another case, *ibid.*, in which, however, a cyst occupying the same site and attended with amaurosis was probably the remains of a blood clot.

74. *Hæmorrhage, hemiplegia, amaurosis, old clot near the thalamus.*

Decaufflet, æt. 68, had an attack of right hemiplegia five years before admission, and his left eye was dim. He was admitted for bronchitis, and died in a few days. Atrophy of the left disk was seen with the mirror.

‘Autopsy. Heart hypertrophied.

‘Encephalon. In the left hemisphere, just above the thalamus, a portion of the small brain is “molle-celluleuse,” and of an ochrey yellow colour. There was no cavity. It seemed to be the remains of a hæmorrhage cicatrised and coloured by altered blood. The optic nerve is atrophied up to the chiasma. The cerebral arteries are ossified.’

Bouchut, ‘Diagnostic des Mal. d. Système nerveux par l’Ophthalmoscopie,’ p. 208.

(No mention is made of the right nerve or disk, and I very much doubt the connection of this unilateral atrophy with the hæmorrhage. More details should have been given concerning the date of the loss of vision, and some other points. T. C. A.)

75. *Hæmorrhage into brain, hemiopia.*

Zagorski gives an account of a female patient, æt. 37, who presented herself at the Eye-clinic in Basel with hemiopic deficiency on the right side in both eyes, and left facial paralysis and hemiplegia. The attack came on suddenly with unconsciousness eight days before, and was clearly a hæmorrhagic effusion into the right hemisphere. The deficiency in vision was completely restored in a few weeks.

‘Monatsbl. f. Augenh.’ October 1867, pp. 322–325.

Several cases of this kind are on record. There is one, for example, in Gräfe’s well-known lecture in the ‘Klin. Mon. Zehender,’ of a man, æt. 68, who suffered from apoplexy with left hemiplegia and hemiopia. The right half of each disk was found to be atrophied and retracted three years after the event, though five months after it no change in the disks could be discovered.

76. *Clot in cerebellum, amaurosis.*

‘A girl, æt. 18, ten years before her death, had an attack of apoplexy, the result of which was amaurosis without any other

paralysis, and habitual headache. An apoplectic cavity of old standing was found in the right lobe of the cerebellum.'

Andral, 'Clin. Medicine.' Quoted by Dr. Hughlings Jackson, 'R. L. O. H. Reports,' iv. 18.

77. Optic atrophy, 'sick headaches,' past history of cerebral disease and fits, also of transient hemiplegia.

Margaret S —, æt. 53, consulted the author at the Dispensary on March 2, 1868, for sick headaches. She did not complain of her sight, nor did she allude to any more serious cerebral symptoms. Had I not fortunately examined her eyes, I should not have suspected anything more than neuralgia, and the case is published as a warning. The attacks of headache occur in one or other temple, and are preceded by a 'swelling of the vein (temporal artery, T. C. A.) as big as her finger.' They recur about once a month, are attended with some nausea, and last about twenty-four hours. They generally set in at about 2 a. m.

O. S. Both disks are atrophied, the more central parts being greyish brown, and the grey white atrophy invading from the circumference. Admits that her sight has grown worse of late.

On further inquiry I find that she has been accused of failing memory. There was some little want of facial symmetry, the left side being a little the stronger. There is slight ptosis of right eye; orbital muscles and iris normal. There is no evidence that this is recent². At the age of 14, had 'water on the brain with inflammation,' for which she was treated by Mr. Garlick. Had fits occasionally, ever since, until the change of life, about three or four years ago. Has had none since. Many years ago two of the fits left right hemiplegia, which lasted three days, and did not affect the speech.

78. Optic atrophy, probably preceded by subacute neuritis, previous history of hemiplegic attack.

Hannah L —, æt. 38, applied to the author by accident at the Leeds Dispensary, instead of going to the room of his surgical colleague. Complained on application (Feb. 17, 1868) of defective vision, which had begun to give her much anxiety. Her right eye said to be the worse.

² I have noticed some ptosis and loss of facial symmetry in several old sufferers from neuralgia. T. C. A.

O. S. Right disk much whiter than left disk and decidedly atrophic. Left disk whitish and rather blurred. Vessels very abundant.

This person applied entirely on account of her eyes, and it was only after a somewhat severe cross-examination, in which, however, all leading questions were avoided, that the following details were obtained. The case, like the former and the following one, is useful only as an example of the difficulties of medical ophthalmoscopy.

Three years and a half ago suffered from pain in head for about six months. She then recovered, and three years after was again in perfect health, and sitting in her chair one evening, when she felt the left arm grow suddenly 'numb and weak,' so that her market-basket fell out of her hand. She tried to rise from her chair, and then fell all her length upon the floor. Says she did not lose consciousness, and tried 'to sam herself up' but could not, and her husband had to raise her. She was then hemiplegic. The leg recovered in a day or two, but the arm remained weak and the fingers 'drawn up' for perhaps two months. She then completely recovered, and has since been in good health. So that it is only by an effort of memory that she recalls the above facts.

79. *Amaurosis first unilateral, then bilateral, subsequent cerebral disease.*

This case is recorded for two reasons : first, to show, what I have said repeatedly in the body of the work, that atrophy of the optic nerve is often a forerunner of other symptoms of encephalic or spinal disease. MacCarthy was for a few months a porter at the Leeds dispensary, and then was suffering from atrophy of the left optic disk. The unilateral character of it was curious, and he was therefore seen by Mr. Teale, Mr. Oglesby, and other observers. It was not until months afterwards that the other eye went in the same way, and now he began to have occasional 'bilious attacks,' headache, and vomiting. These, however, had so little unusual in them, that, interested as I was in the case, I should not have discovered this had he not been one day absent from his duties on account of an attack. Shortly after he left the dispensary, and a year or more must have elapsed before I espied him in the medical wards of the infirmary, under Dr. Heaton's care for manifest encephalic disease. He was suffering under considerable paralysis

of all four limbs, and had headache and vomiting. The case is suggestive of cerebellar disease; but I need not enter into this, as there is as yet no autopsy, and the point of interest—the long antecedence of amaurosis—is established.

80. *Aneurism of anterior cerebral artery.*

A man, æt. 37, became suddenly insensible while at work, but quickly recovered and resumed his work. In three weeks had another fit, and remained in stupor three or four days. Seems dejected, sallow, and morose, and complains of vertical headache made worse by stooping. Pupils dilated but act under light. Some days afterwards became suddenly comatose and stertorous, and died next morning.

Autopsy. Extravasations of blood in various parts of encephalon, and an aneurism of the right anterior cerebral artery the size of a hazel nut was found pressing upon the right optic nerve. It had burst into the lateral ventricle.

As there was cataract of the man's right eye its blindness cannot be referred to the aneurism, but the case is useful as showing how such an aneurism may affect the optic nerve.

Abbreviated from Mackenzie, third ed. 1840, p. 877.

81. *Tumours of anterior lobe, meningitis, amaurosis, autopsy.*

Mrs. E. P. of Barnsley. Admitted into Leeds Infirmary under the author on Aug. 6, 1870.

Family history good.

Personal history good till three and a half years ago, when dull, aching, constant headache and morning sickness set in. Sight has been affected for about nine months, and has lately become much worse. She is now indeed almost blind, and has white atrophy of both nerves. A general weakness of the limbs without definite palsy came on at the same time and now continues. On admission she is incoherent in ideas, and jumbles words. She speaks very slowly, but is scarcely intelligible. Died Sept. 18.

Autopsy. Two small superficial tumours, about the size of beans, in the anterior lobe of the right hemisphere. Brain substance around them much degenerated; evidences of proliferation slight save on the surface. There is adhesive meningitis all over the anterior lobe both above and below. All the membranes are

adherent to the orbital plates below, and the adhesions extend as far as the chiasma, the loss of sight being due no doubt to an adhesive basilar meningitis.

82. *Glioma of left anterior cerebral lobe, double optic neuritis.*

‘A man, æt. 23, under the care of Dr. Ramskill, had convulsive attacks; he suffered intense pain in his head, and there was double optic neuritis. It was almost certain from these symptoms that there was intra-cranial tumour. He was doing very well, being about the ward. He had a good appetite, and was able to read, when one night he was seized with a convulsion, became very deeply comatose, and died in two or three hours. Dr. Sutton found at the autopsy a gliomatous tumour of the fore part of the left anterior cerebral lobe, with recent effusion of blood to the extent of several ounces.’

Hughlings Jackson, ‘Lancet,’ Oct. 23, 1869.

83. *Case of hydatid cyst in the right cerebral hemisphere.*

(Manchester Royal Infirmary, under the care of Dr. MORGAN.)

‘The subject of this case was a little girl, æt. 7, who was admitted into the Manchester Royal Infirmary on November 22, 1869, and died February 1, 1870. At the time of her admission, it appeared that the patient had been ill for nine months, her illness commencing with convulsive seizures. These seizures were succeeded by paralysis of the lower extremities; the pupils were both considerably dilated, though there was no paralysis of the muscles of the eyeballs on either side. Her sight was much affected; there was no apparent loss of cutaneous sensibility. During the last four weeks of her life she was comatose; she became gradually weaker, and died February 1, 1870.

‘*Inspection sixty hours after death.*—On removing the calvarium, nothing unusual was observed; but, as soon as the dura mater was detached, a considerable portion of the cyst was seen lying immediately below it in the right cerebral hemisphere. On removing the brain from the skull, this cyst readily slipped out from the cerebral hemisphere, without discharging any of its contents. The cyst had occupied the anterior and middle lobe of the hemisphere, extending inwards to the lateral ventricle. A considerable portion of the corpus striatum and part of the optic thalamus were absorbed from pressure of the cyst. There was no softening or dis-

ease of any portion of the brain surrounding the cyst. There was no fluid in the right lateral ventricle; but that on the left side contained about an ounce of clear serum. In other respects the brain was perfectly healthy. The cyst weighed $18\frac{1}{2}$ ounces, and contained 18 ounces of clear fluid. The fluid had a faint alkaline reaction. Specific gravity, 1011. Under the microscope numerous echinococci and detached hooklets were founded.'

'British Medical Journal,' June 18, 1870.

84. *Tumour of middle lobe, neuritis, autopsy.*

An encysted tumour about the size of a mandarin orange, and weighing three and a half drachms, was shown to the author lately by Mr. W. N. Price. It had been removed from Miss F., who during life had suffered from anæmia, and from much neuralgia in the head and face. She had also transient numbness of the right side, with giddiness, some weeks before death. The only marked symptom was blindness, which followed optic neuritis of both eyes. The notes of the autopsy are very inadequate. The tumour was found in the upper and outer part of the left middle lobe of the cerebrum; 'effusion-like lymph' surrounded it, and the brain substance was extensively softened. The tumour did not seem to be truly encysted, but was like a mass of old tubercle.

A brother and a sister had died of tuberculosis.

85. *Headache, vomiting, convulsion, optic neuritis, hydatid of left hemisphere.*

R. B., æt. 8, had been ill eight or nine months with occasional headache. After an interval of some weeks the headache returned with great severity, generally coming on at 4 a.m. and remitting at 10 a.m., when there was vomiting. Weak sight six weeks before observation on April 9, 1867. Convulsions a month ago, and occasionally since; obvious but slight weakness of right arm, side, and leg. Died in stupor, April 26.

O. S. April 23. Disks dirty white, swollen; veins irregular, partly buried in swollen disk. Disks no real boundary, gradually melting into fundus. (Ischæmia.)

Autopsy. Cyst in posterior half of left hemisphere, lined by gelatinous firm wall, one fifth of an inch thick. Wall tore easily, and was separable from brain substance. Lateral ventricles 'dilated' (with fluid?) and bones of skull tending to separate.

Cavity extended forwards to middle of ascending parietal convolution and backwards to end of outer wall of posterior cornu.

Abbreviated from Hughlings Jackson, 'London Hosp. Reports,' vol. iv. p. 391.

86. *Blow on head, tumour of right hemisphere, atrophy of disks.*

Mr. Handcock has been so kind as to forward to the author the following notes of this case :—

'I fear I cannot give you very ample details in reference to the boy you saw with Mr. Teale and myself in Accommodation Road. His name was John S., æt. 10½ years, and he died on September the 16th, 1867, one year and seven months after receiving a blow with a stone on the right side of the head. He had always enjoyed perfect health before that time ; nor is there any hereditary tendency to scrofula in the family. Soon after the blow he began to complain of pain in the part where he was struck, and in a few weeks the headache became at times so severe as to cause him to shriek with the severity of the pain. These severe attacks of pain came on every two or three weeks, were attended with vomiting, and generally lasted a day or two. In the intervals he was able to walk about, but was observed to drag the left leg. He then gradually lost the sight of the right eye, and the pupil became fully dilated. During the last nine months of his illness he became hemiplegic on the left side, lost power over his bladder, and became totally blind. His hearing was preternaturally sharp, and his memory was not much impaired.'

When seen by Mr. Teale and myself, the optic disks were quite atrophied. We found a tumour, a loosely-built sarcoma, in the posterior lobe of the right hemisphere. It was about the size of a pigeon's egg. The brain around for a short distance was softened, but the rest of the encephalon and the membranes were healthy.

87. *Tumour of base of encephalon, blindness of right eye, autopsy.*

This case is related by Dr. G. A. Rees in the fourth volume of the 'Pathol. Transactions.' It cannot be cited as an instance of unilateral optic change, as the mirror was not used. The right eye was blind, and the state of the left is not mentioned. It is a remarkable instance of the way in which such tumours grow and involve successive parts. It was pear-shaped, about 3" long, and ¼" at its greatest diameter. The right optic nerve was lost, the

left was adherent to the side of the tumour and considerably diverted. It seems, from the rigidity of the arm observed at the outset, to have commenced near the right crus cerebri, and as it extended itself forwards so it involved the optic nerve, the third pair, the right olfactory lobe (also gone), and the grey matter of the right anterior lobe, as was marked by loss of vision, strabismus, and subsequently impaired intellect.

88. *Tumour of crus cerebri, double optic neuritis.*

A boy, æt. 9, was under Dr. Hughlings Jackson's care at the London Hospital, for paralysis of the third nerve on one side, and hemiplegia on the other. These symptoms had come on slowly with headache; and there was a double optic neuritis. He died during the night, of effusion of blood from a tumour of the crus cerebri, which caused the paralytic symptoms. The palsy of the third nerve on one side, and of the arm and leg on the other, pointed to disease of the crus cerebri. The gradual onset of the paralysis, and its complication with optic neuritis, made it certain that the disease was of some *coarse* kind. The age of the patient rendered it most probable that this coarse disease was tumour. His sudden death led to the inference of hæmorrhage from tumour.

Hughlings Jackson, 'Lancet,' Oct. 23, 1869.

N.B. I have been so fearful of leaving my main subject, that I have not discussed points in diagnosis of tumours which were not ophthalmoscopic. I may say, however, that I have twice seen sudden coma and death following some convulsions in cases of simple tumour without hæmorrhage. Such a case, for instance, is recorded in the 'Catalogue of S. Geo. Museum,' No. 187, p. 392.

89. *Tumour of corpora quadrigemina, amaurosis.*

'E. S., æt. 27. Amblyopia one month with headache, both eyes being affected. She could only count figures close at hand. The disk was greyish and very prominent, its margin invisible. Veins moderately congested. Iodide of potassium ordered. In a short time she was blind, and in a few weeks vomiting, voracity, constipation, and dysuria set in, with attacks of loss of consciousness and slight twitchings. There was gradually augmenting deafness, and towards the end she was violently delirious. She died four months after admission. The state of the pupils is not mentioned.

'Autopsy. Great and general congestion both of the sinuses and

of the brain. A neoplasm extends from the corpora quadrigemina into the interior of the pons varolii. The region of the corpora quadrigemina consists almost entirely of connective tissue; this proliferation has no distinct limits where it extends into the pons. Scattered through the neoplasm are the elements of tubercle.'

W. Wagner, 'Klin. Monatsbl. f. Augenh.' 1865, p. 159; quoted in 'Ophth. Review,' ii. 404.

90. *Amaurosis with dilated pupils, general palsy, tumours of corpora quadrigemina.*

'A man, æt. 30, had complained for a year of severe headache, loss of memory, dimness of both eyes, and faintness. Aspect cachectic, dull in reply, pupils much dilated and sluggish, all objects appear misty. Gait feeble, and is soon wearied, hand-pressure weak, drowsiness alternates with twitchings of limbs and objectless cough. In a few days sank into a stupid state, and died completely paralytic.

'Autopsy. Inner membranes infiltrated with serum, convolutions flattened, ventricles distended. In corpora quadrigemina a medullary growth about the size of a nut, which separated the thalami from each other and sent a small conical process into the fourth ventricle.'—Rosenthal, 'Nervenkrankheiten,' p. 83.

91. *Tumour of crus cerebelli ad pontem, neuritis, autopsy.*

A man, æt. 37, came under Mauthner's care with a fully developed neuritis in both eyes ('in beiden Augen das vollkommen entwickelte Bild der Neuritis'). His sight was tested and found to be perfect. This man enjoyed full acuteness of vision ('dieser vollen Sehschärfe erfreute') until the end of his life. He died suddenly. A tumour was found (a sarcoma of the size of a walnut) in the right crus cerebelli ad pontem, together with a considerable dropsy of the ventricles. Papillary neuritis was found, with thickening of the connective tissue, the layers of the retina up to the papilla being normal.—Mauthner, 'Lehrbuch,' p. 293.

92. *Tumour of cerebellum, ischæmia papillæ, venæ Galeni turgid.*

Dr. Simpson, of Manchester, read notes of a case of cerebral disease of much interest. The patient was a boy aged 14, who, up to the end of 1869, had enjoyed good health, and whose family

history was satisfactory. About Christmas, he began to complain of severe shooting pain at the back of the head, intermittent in character, and at times so severe as to make him scream out. Soon afterwards he began to vomit; at first in the morning, but subsequently also after his meals. The vomiting was unaccompanied by nausea. His bowels became very costive. He continued in this state for some time, when, about the middle of April, he began to have a dull, heavy expression, and also showed some difficulty in walking and articulating. It soon became evident that his sight was becoming impaired, and though he remained fairly intelligent, his memory became very defective. There was slight paralysis of the internal rectus of the left eye. The symptoms gradually became worse. His gait was not that of paralysis, but of want of co-ordinating power.

O. S. On July 25, his eyes were examined with the ophthalmoscope by Mr. Windsor, who reported a large and swollen condition of the optic disk, with no distinct boundary, a swollen and tortuous condition of the veins, and other evidence of intracranial pressure. His progress was steadily downward, and he died on September 17, comatose. At the *post mortem* examination, the head only could be examined. The veins of the dura mater were turgid, and the visceral arachnoid rather thick and opaque; and there were several ounces of slightly turbid cerebrospinal fluid. There was a little lymph about the optic commissure and anterior margin of the pons. The venæ Galeni were very turgid. The whole brain substance was softened, particularly the parts at the base. Both lobes of the cerebellum contained numerous yellow masses of tubercle, varying in size from a pea to a marble. No tubercle was found elsewhere in the brain, and during life there was no evidence of its presence in any other organ. Dr. Simpson referred to the question of diagnosis, and discussed particularly the differential diagnosis of tubercle and hydatid cyst of the brain.

‘British Med. Journal,’ Oct. 22, 1870.

93. *Palsy of recti externi, neuro-retinitis, death, tumour of pons.*

‘Franz Kothasek, typesetter, æt. 38, admitted into Oppolzer’s wards on Feb. 13, 1865. Headache some years. Severe headache in forehead, temples, and occiput for seven weeks. Vomiting

for eight days, amblyopia, which set in suddenly, for five weeks. Right eye quite blind, left highly amblyopic.

‘Dizziness prevents his standing. Pulse 135.

‘O. S. (by Dr. Rydel). Bilateral neuro-retinitis, with numerous oval hæmorrhages, especially in right. Disks swollen, turbid, blurred. Both external recti palsied.

‘Improved on galvanism of the sympathetic.

‘In the summer and autumn returned with headache, and tubercle of lungs and larynx. Weakness of extremities without definite palsy came on, and death took place on December 23.

‘Autopsy. Besides tubercle elsewhere was found in the *head*—calvaria thick and compact; dura mater stretched, inner membranes rather infiltrated with serum and easily removed. Brain somewhat moist and firm. Lining of ventricles thickened and half an ounce of serum in the cavities. In the lower half of the left anterior portion of the pons a round firm cheesy tubercle the size of a cherry. The lump had penetrated to the base, and had grown from the deep transverse layer of the pons and pressed aside the strands of the pyramids without destroying them.’

Benedikt, ‘Elektrotherapie,’ pp. 257, 258.

94. *Railway accident, changes in the fundus, amblyopia, spinal injury.*

Mr. C. was seen by the author in consultation with Mr. Teale of Leeds, the late Dr. Garlick of Halifax, and Mr. Hiley of Elland, on Jan. 18, 1868. In March 1867 he had received a concussion in a railway accident, which caused some unconsciousness and some injury to the spine. Soon after, his sight, which was of much importance to him in business, began to fail. When we saw him he had fever, great dizziness, some delirium, some incontinence of urine, sexual impotence and priapism; also severe shooting pains in the legs, which however were not palsied. He had two definite and acutely tender points in the back, one about the seventh cervical vertebræ, the other over the last three lumbar vertebræ.

O. S. Pupils equal and sensitive to light. Reads large primer with difficulty. Fundus in both eyes very much altered. There is no trace of a disk, the whole of this region being of a reddish injected appearance, the red in the right eye having a daffodil tint—a sort of yellowish pink. The retinal veins were full and dark, and the arteries, as such, were indistinguishable.

95. *Railway accident, spinal injury, vascularity of disks.*

Mr. L — was a patient of Mr. Seaton of Leeds, and was seen likewise by Mr. Teale and the author. The case was a most interesting one, and will I hope be published by Mr. Seaton. It is only to the point at present to say that when we saw him together he was utterly paraplegic both of motion and sensation, and in daily fever. The cerebral functions were also much disturbed, hallucinations and delirium being very frequent. There were the two tender points on the spine, which are so commonly found in these cases, namely, one in the lumbar, the other in the lower cervical and upper dorsal region. I saw him two months after the accident.

O. S. Both retinas were hypervascular, the disks were slightly veiled by exudation, and their edges thereby also a little dimmed.

96. *Railway concussion, amblyopia, suffused disks.*

Mr. S. was seen by Mr. Teale and the author on the 23rd of August, 1866, and the fundus was drawn by Mr. Aldridge.

Five weeks before, he had a concussion in a railway accident, but did not seem to be much hurt at the time. He believes now that his back was struck, and it is very tender to pressure over the sixth and ninth dorsal vertebræ. He is now depressed, confused in mind, and easily fatigued. After talking to us for half an hour, his words run into one another.

His sight, excellent before the accident, is now dim. He cannot read No. 1 Jäger, but reads No. 3 with difficulty, aided by a No. 20 convex lens. Soon after the accident his pupils were dilated and sluggish. At present the left pupil is normal, but the right dilated, and acting slowly through the medium of the left.

O. S. Right eye. Disk suffused, no margins visible, some œdema about and upon the disk, veins of retina tortuous, those passing downwards very tortuous.

Left eye. As right, except that the vessels of the retina, though full and dark, are not tortuous.

I never saw this patient again.

97. *Injury to neck, paraplegia, hyperæmia of optic disks.*

S —, of Kippax, came to the author at the infirmary, complaining of palsies which had followed a blow on the head. Finding that the palsies were spinal, it was soon made out that the blow on the head was the result of a heavy fall upon it, the fall

severely twisting and spraining the cervical vertebræ. Over the fourth and fifth vertebræ there was thickening and much tenderness, of which he was aware, but had not supposed it to be of importance. He was weakened in arms and legs, and had abnormal sensations. There was occasional priapism and vesical derangement. The case closely resembled a common kind of railway injury.

O. S. About five weeks after the injury both disks became hyperæmic and somewhat suffused, the whole of this part of the fundus being of a uniform yellowish red. The disks were indicated by the convergence of a number of new small veins. The field of vision was not contracted, but perception was decidedly diminished.

98. *Caries of the spine, cerebro-spinal meningitis.*

The next autopsy was on the body of a female who had been for many months in the hospital suffering from psoas abscess. On opening the head, the arachnoid appeared greasy, and a quantity of pus was seen running along the upper part of the hemispheres; and there was a quantity of puriform-looking lymph under the pia mater at the base of the skull between the optic commissure and the medulla oblongata. The brain-substance was firm, excepting the parts forming the lateral ventricles, the fornix, the septum lucidum, and the surfaces of the corpora striata and optic thalami, which were softened; and there was an excess of fluid in the lateral ventricles. The softening of these parts, and the quantity of lymph at the base of the brain beneath the pia mater, looked, at first sight, like tubercular meningitis; but the pus seen running on the lateral parts of the hemispheres were unlike tubercular disease. The membranes were carefully examined, and no tubercle was present. As there was no disease of the brain-substance or tubercle in the membranes to account for the pus being beneath the pia mater, it was suspected that the disease had extended upwards from the spinal canal. On examining the spinal cord, a quantity of puriform lymph was seen extending from the lumbar region to the base of the brain, and the bodies of the lumbar vertebræ were in the condition known as caries, and in the substance of each psoas muscle was a collection of pus.

About two days before this patient died, she complained of great pain across the forehead, and vomited; delirium of a very active kind set in; there was retention of urine; the patient was very restless, and moved her arms and legs about a great deal. Con-

sidering the great amount of disease all around the cord, it is interesting to note that there was no marked or complete paralysis: at the same time, it must be remembered that the surface of the cord only was affected, and that its substance was not softened.

‘British Medical Journal,’ July 2, 1870.

99. *Spinal disease (caries), hypercæmia of disks.*

C. S., æt. 37. Admitted under the author’s care October 16, 1868. Three years ago, when in health, was struck with a brush handle on back of neck. Some few weeks after, much stiffness was noticed, and intense pains striking down into both shoulders and arms and up the occiput to the vertex. A tumour was then said to have appeared at the back of the throat, which caused difficulty in swallowing and regurgitation of fluids through the nose. This swelling ‘burst’ and disappeared. He remained some weeks under observation, during which time the arms became much palsied and the legs rather weak. He had intense occipital and vertical headaches, with vomiting. There was thickening and tenderness over the middle cervical region, and he referred the pain of movements to this part. He obtained little benefit, and we lost sight of him after about five months, his state being decidedly worse than on admission.

O. S. During all this time both disks were highly hyperæmic and troubled. Margins invisible. Retinal veins large. No marked prominence of disks. There was some deficiency of visual power with small types. The right disk was always rather worse than the left. Pupils a little dilated.

100. *Spinal disease, with optic signs.*

B. B. A——, æt. $3\frac{1}{2}$, 12 East Street. Leeds Infirmary, April 13, 1868. Pale, weakly child, with somewhat scrofulous history. Six months ago an enlargement appeared in the lower cervical region of spine. There is now great distortion there, bringing the third and seventh cervical spine up to the occiput. There is much neuralgia, and some weakness of all four limbs. Has one or two enlarged glands under neck.

R. Veins decidedly large, but not very dark, and the disk looks misty. The edges can however be made out.

L. As R., except that edges of disk cannot be seen. The disk is not however much swollen. There is a decided amount of light

suffusion without any very dense opacity over a wide belt of retina.

The sight is reported to be decidedly affected ; the child thinks there are 'clouds in his eyes,' as his mother says. But he can distinguish objects well enough.

101. *Injury to nape, disease of vertebræ, palsy of sympathetic nerve, ophthalmic signs.*

Johnny — was long a favourite patient under the author's care, and his symptoms were most interesting. Struck by a big boy upon the 4-6 cervical vertebræ, his strumous habit was unable to repair the mischief, and caries set in. Paraplegia of arms and legs and bladder then appeared, and the body wasted below the seat of injury. Some weeks after appeared also palsy of the left cervical sympathetic. This depended upon the pressure of morbid products, and varied therewith. During three weeks it transferred itself to the other side, the left side symptoms disappearing. These on either side were heat of cheek and ear, often amounting to an excess of 5° or 6° (C) over the other side, hyperæmia of the same region, and undilateable pupil. A month before the appearance of these changes, the ophthalmoscope had showed injection and slight suffusion of the disk and neighbouring retina. These appearances remained constant, and showed no variation with the inconstancy of the heat and hyperæmia of the neck, cheek, and ear.

102. *Myelitis, subsequent atrophy of disks.*

Christopher B., æt. 26, admitted under the author's care on Nov. 2, 1866. He was a long time under notice, and the case excited much interest, as he undoubtedly suffered from severe acute myelitis, with bed-sores and complete paraplegia, and yet recovered. The bed-sores began to heal about Dec. 4, and during the many following weeks he slowly regained power. As he regained power his optic disks began to whiten and the vessels to wane. This atrophy, however, seemed not to progress into the third stage, not, that is, during the few months he remained under observation. The field of vision was lessened decidedly, but central vision remained fairly good. He was unable to read the newspaper, however, for more than a few minutes, and he had some teasing scotomata. We lost sight of him about the middle of 1867.

103. *Chronic disease of the cord, atrophy of optic disks.*

R. B. was sent to the author by Mr. Sedgwick, of Boroughbridge, on June 3, 1869, complaining of paraplegia. It was incomplete, but sensation and motion were both decidedly impaired. His symptoms set in about twelve months before. He now reeled in his walk, and the bladder was partially palsied. There were no special symptoms of locomotor ataxy.

O. S. Atrophy of optic nerves in both eyes. Central vision somewhat impaired, and visual field contracted. Vessels small and waning. No evidence of foregone neuritis, acute or chronic.

Improvement of both sight and legs under careful Faradisation and the use of iodide of potassium.

104. *Paraplegia, hyperæmia of disks.*

Ann C——, æt. 50, admitted under me on Sept. 4, 1868. Debility for two years, and some loss of appetite and flesh in consequence of a severe fall, which hurt her back in upper dorsal region. No local weakness till four months ago, when numbness and pricking with weakness invaded the legs, and extended to the lower part of the trunk. At the same time shooting pains down back and legs, like those of ataxy, set in; they also passed down left arm. On admission, there is a point of the spine about the first two dorsal vertebræ which is acutely tender on pressure. No distortion, unless some prominence of the spines. There is decided weakness of both legs, though she can stand; also of left arm. Sensation to touch deficient; feels as if walking on wool. No marked ataxic symptoms. Left arm feels 'as if covered up.'

O. S. Examination on admission. Right optic disk suffused and pink, edges invisible, retinal vessels natural in size. Left optic disk as right, but not quite so much disordered. Pupils not contracted.

O. S. Examination three months later. Disks have been gradually changing from red suffusion to greyness. Edges still invisible. Vascularity less. Now there is weakness of both arms, and frequent spasmodic contraction of left arm. She left the house, and has since disappeared.

105. *Locomotor ataxy, chronic optic neuritis.*

Jane F——, æt. 28, was admitted into the Leeds Infirmary in January, 1868. She has lancinating pains and loss of sensation in

the feet and lower legs, as tested by the compasses, &c. She feels as if she were walking on cushions, and is scarcely conscious of any inequalities in the ground on which she walks. She loses her legs in bed, especially if they be crossed. She can walk with fair readiness and speed, if she may look at her feet, and may run the tips of her fingers along the wall. Put her feet together and she totters; close her eyes and she falls. There is little or no loss of muscular power. She says she has no loss of vision whatever, nor does she seem to have on testing her with types. The pupils are a little contracted. On dilatation, I found the disks whiter than natural, the veins a little larger, the arteries smaller, and decided marks of exudation both on the margins of the disks and dotted closely around them. Mr. Teale kindly verified this observation for me.

106. *Neuralgia, with evidence of disturbance of cervical sympathetic, disks normal.*

Mrs. M. A. J., æt. 26, came under the author's care in June, 1868. For five years has been subject to the exhaustion of bleeding piles, which are now cured. Three years ago suffered also from an attack like the present. Four months ago was confined, and is suckling the baby. She now suffers from very intense pain in all branches of the fifth nerve on the right side. When the attacks come on she has (as we have many opportunities of seeing) a contracted pupil, and she sweats profusely (to her own great annoyance) on the right face, neck and shoulder, so that water runs off her and wets all the clothing in this region. The sweating and pain are generally synchronous, but not always. The right vocal cord is also paralysed, as seen with the laryngoscope. Under the use of hypodermic morphia and of quinine, with iron and valerian, she improved much, the pain departing first. A numbness of the right side of the face outlived the pain for some weeks, and the unilateral sweating also occurred occasionally without pain for some time.

O. S. The disks, dilated with atropine, were repeatedly examined, both during the paroxysms and between them, but never detected anything abnormal, unless it were some degree of persistent anæmia, which was visible also in the mucous membranes and elsewhere.

107. *Scarletina, Bright's disease, retinitis, recovery.*

‘A lad, æt. 14, had suffered from scarletina, Bright's disease, and

retinitis six years before; at that time the visual power was only quantitative. A year later he appeared healthy, and by means of weak convex glasses was able to do school tasks. No change in vision has occurred during the last four years. He is now a powerful athletic boy, and the heart and urine are normal. The visual field is normal. The remaining amblyopia, which was accurately noted, was accounted for by the dull white colour of the disks, which are indistinct; around them is a dullish white ring with some white patches at its edge. The rest of the fundus is normal.'—Höring, quoted in the 'Oph. Review,' i. 159.

108. *Scarletina, albuminuria, retinitis.*

(Under the care of DR. RUSSELL, of Birmingham.)

'A man, æt. 34, was admitted February, 1869, with symptoms of four months' duration, dating from an attack of scarletina. He continued under care till the end of September, when he left improved. His symptoms were, anæmia, considerable anasarca, with tendency to effusion into the cavities. The urine was copious, at times reaching seventy ounces; specific gravity 1012-20; the albumen from one-fifth to two-thirds the bulk of the urine. The urine presented sometimes intracellular transparent casts; sometimes a copious deposit of fibrinous casts of small and full size, with numerous fat-cells. At his admission he read Jäger's No. 1 brilliant.

O.S. 'Examination, March 20th.—The retina was bluish white and œdematous. There were several small glistening specks around the yellow spot. The veins were tortuous and swollen.

'April 24th. The changes in each eye had advanced considerably, especially in the left, where the disk was scarce distinguishable, except by the entrance of the vessels. There were a few minute hæmorrhages, and white glistening spots, chiefly around the yellow spot and the entrance of the optic nerve.

'May 20th. The outline of the left optic disk had cleared, but that of the right eye was so obscure, that it could scarcely be made out. There were blood-specks and white spots.

'Sept. 13th. Both disks were obscure; the veins full and tortuous. The left retina was so much flecked with small glistening white specks as to seem dappled. Here and there were traces of old extravasation.'

'British Medical Journal,' January 15, 1870.

109. *Albuminuria, retinitis.*

Edward Thomas H——, æt. 40, was admitted, July 1867, under Dr. Johnson, with Bright's disease. He applied on October 24 to Mr. Soelberg Wells about his sight, which had begun to fail for about two months. On examination it was found that it was greatly impaired, for with the right eye he could only read No. 20 of Jäger's test types, and with the left No. 19. A strong convex lens (No. 5), enabled him to read No. 6 indistinctly with the right eye, and No. 4 with the left. The field of vision was relatively good in each eye. The refracting media were transparent. The ophthalmoscope revealed the existence of well-marked nephritic retinitis. In the right eye, the optic disk was somewhat opaque and indistinct, and its outline irregular and ill defined. The opacity of the disk was due to an inflammatory infiltration, which extended to some distance (three or four times the diameter of the disk) into the retina; the latter being here also studded with numerous irregular white patches and dots, more especially towards the region of the yellow spot. At the latter point were seen the peculiar brightly-shining stellate spots, which are so often observed in the retinitis of Bright's disease. The retinal veins were dilated and tortuous, but not to a very considerable extent, whereas the arteries were markedly attenuated. Numerous small striated blood effusions were strewn about the retina and optic disk. The condition of the left eye was very similar in appearance, excepting that the peculiar white stellate dots in the region of the yellow spot were absent, and the extravasations of blood were not numerous.

Note by Dr. Johnson.—'This patient had general dropsy; the urine contained a large amount of albumen and numerous oily casts and cells. I have no doubt that the case was one of large white fat kidney, but the man left the hospital shortly before his death, and no p. m. was made.'

Communicated to the author by Dr. Johnson.

110. *Albuminuria, retinitis, autopsy.*

(Under the care of DR. RUSSELL, of Birmingham.)

'A man, æt. 29, first came under notice in January 1867, when his symptoms were of six months' duration. They were not permanently relieved till the end of the year, and again manifested

themselves in October 1868, after a period of fair health. He was again admitted in January 1869, and died on June 2. He had epistaxis at an early date in his disease, and again the day after his second admission in January 1869. He first observed his sight to be impaired at the end of 1868. His work, being of a very delicate character, afforded him a fair test. He found that the point of the pens on which he was operating looked twisted. It however appeared that faulty accommodation was concerned in the defect, as his surgeon, Mr. Figgins, always found his pupils much dilated, and was able to improve his patient's vision by employing the Calabar bean. The same condition of pupil was apparent at his admission, with very defective sight.

O. S. '*Examination*, January 21st, 1869.—The outline of the disk was indistinct; the vessels diminished in number; the veins rather full. There were many small patches of extravasated blood in the superficial and deep layers of the retina. Shining white spots, irregular in size and shape, were scattered over the retina, and there were also patches of degeneration.

'*Examination*, May 20th.—There was hæmorrhage beneath the conjunctiva of the right eye, covering half the globe. The optic nerve was whiter than natural. The arteries were small; the veins full. The white glistening specks were especially abundant around the yellow spot.

'*Autopsy*.—His kidneys presented a rather early stage of the granular fatty degeneration of Johnson, presenting single coils of tubes filled with oil, and hypertrophy of the small arteries.'

Dr. Russell adds in a private letter to me:—

'*Birmingham, January 22, 1870.*

'DEAR DR. ALLBUTT,—I was unfortunately prevented from being present at the post-mortem, but received the following particulars: "Kidneys somewhat larger and heavier than natural; capsule peels off easily, surface smooth, pale fawn coloured. Heart—left ventricle thick, pale fawn coloured; cavities of both ventricles dilated." I am sorry that they neglected to weigh the organs.'

111. *Granular kidneys, hypertrophy of left ventricle, arterial degeneration and dilatation, retinitis.*

Mrs. A., æt. 46, a patient of Mr. S. Hey. Noticed one day, after some extra-exertion, that she had a pulsating swelling in the

neck. I was requested to see her in consultation with Mr. Hey. She had a very sallow aspect, was ill-nourished, skin harsh and wrinkled, urine abundant, of a low specific gravity, and containing small quantities of albumen. A pulsating tumour was present in the right neck above the collar-bone, and its percussion dulness extended downwards over the upper third of the sternum and for a finger's breadth to the right of the sternum. The swelling was of very variable size, and greatly receded when perfect rest was observed. It was always present, however, to the extent of a finger's breadth above the right collar-bone. The swelling could also be felt to pulsate above and behind the sternum on pressing the finger into the notch. There was a murmur with the first movement of the heart, and this was heard all over the tumour. Two years' observation of this patient convinced Mr. Hey and myself that we had to do with a diseased arterial system generally, and in particular with a dilated aorta and innominate artery. We did not examine the retinae until one morning we were both summoned to her, because she had become suddenly blind of the right eye. On examination with the mirror we found an advanced state of nephritic retinitis. One disk was surrounded by a rampart of fatty accumulation; in the other eye there were disseminate patches, and a constellation round the yellow spot.

Mr. Teale also saw the patient on several occasions. She suffered from slight cerebral symptoms for some months after this, and ultimately died in about three years from the time of our first visits, worn out by general disease. There were no distinct symptoms of encephalic hæmorrhage, but rather of embolism.

112. *Bright's retinitis, granular kidney, no hypertrophy of heart.*

Thomas W—— was admitted under the author's care in the Leeds Infirmary on the 5th of August, 1869. He complained of general debility and ill health. His complexion was sallow, he was wasted, and his skin was harsh. His urine was of s. g. 1010, and it contained a decided though small quantity of albumen. The valves of the heart were competent, its dulness of normal extent, and its cavities of normal size.

O.S. The eyes presented a marked example of albuminuric retinitis. Both retinae were in advanced disease; hæmorrhages numerous; fatty patches large and confluent. Vision much deteriorated.

The patient ceased to attend after a few weeks.

113. *Retinitis, waxy kidney.*

'Archibald M——, æt. 40, under the care of Dr. Grainger Stewart, was suffering under anæmia, with the symptoms of waxy disease of the viscera of the abdomen. His vision was impaired.

'O. S. Retinitis was observed by Dr. Argyll Robertson in both eyes, with commencing degenerative changes, and several points of blood extravasation.

'He sank and died comatose from anæmia about three weeks later. On post-mortem examination, waxy degeneration of kidneys, liver, and spleen was found.'

From Dr. Grainger Stewart's work on 'Bright's Disease.'

114. *Syphilis, headache, neuro-retinitis.*

S. M., æt. 28, was admitted under the author on Dec. 17, 1869. Contracted syphilis six years ago, the chancres, of which there were two, being hard and difficult of cure. His habits had been any thing but temperate. Four months ago, after a drinking bout, a severe headache commenced in the left temple, 'striking into the left eye.' The sight of the eye gradually failed, and in a week was gone; the other soon followed it. On admission he had intense paroxysmal headache, and could scarcely distinguish light. As I had scarcely entered into the case on my first visit, I ordered a blister over the supra-orbital branch, which was successively painful, with morphia and aconite dressings. He had obtained no relief in three days, when I saw him again, and found a typical example of neuro-retinitis in both eyes. Under full specific treatment he lost his headache, and regained his sight so far as to read the newspaper. Much of the exudation about the disks was reabsorbed before we lost sight of him.

See many cases of syphilis with neuro-retinitis in a paper by the author in the fourth vol. of 'S. Geo. Hosp. Reports.'

115. *Syphilitic retinitis.*

Feb. 11, 1869.

These two cases the author owes to the kindness of his friend Mr. Oglesby, of Leeds.

E. C., æt. 29, single woman, complains of failing sight. Has been ailing for some time from rheumatic pains, chiefly in the head, which were at one time so severe as to produce convulsive attacks of a serious nature. She shuns a bright light, and shrinks from an

ophthalmoscopic examination, as it is extremely painful to her. The outline of the disks is obscure, and the retina in their immediate vicinity is very hazy and grey. The retinal veins are enlarged and tortuous, but the calibre of the arteries is normal. On close examination, minute specks of pigment are found to be scattered over the periphery of the retina, not unlike the deposition of pigment which occurs in cases of night blindness. She has been under treatment for constitutional symptoms, and her general health is much improved, but vision steadily diminishes. Treatment proved unavailing, and when last seen she complained that each day her sight grew worse.

Also

116.

July, 1870.

A young man labouring under the hereditary form of syphilis, and having the characteristic facial expression, applies for relief on account of severe aching pain in the globe of the right eye and supra-orbital region. He also stated that vision had for some weeks been very imperfect. There was slight tension, but no appearance of inflammation of cornea or iris. The ophthalmoscope disclosed an interesting condition of retina. Two patches of lymph of considerable size rested on the retina, one immediately above, the other immediately below the optic disk. Both were in the course of large vessels which crossed the patches, but were never so entirely lost—disappearing at one edge, re-appearing at the other—as we find in congenital nerve patch. The retinal vessels were extremely numerous and much enlarged, though not tortuous. The other parts of the retina appeared healthy. Under appropriate treatment—cod liver oil and tonics—the lymph patches slowly disappeared, leaving behind them functional impairment of retina.

117. *Lead poisoning, optic neuritis.*

‘A young girl presented marked symptoms of lead poisoning; had suffered of late from several sudden losses of consciousness. She was able to distinguish lamplight at two yards’ distance.

‘O. S. Pupil dilated, field of vision much contracted. Media of eye transparent. Disks swollen, presenting a reddish grey and opaque colouring; retina around the disk presents like opacity. Retinal veins large, tortuous, and dark coloured; arteries small and pale.’

E. Meyer. One of two cases published in the ‘Union Medicale,’ No. 76, 1868.

118. *Lead poisoning, amblyopia.*

'A house painter, æt. 35 years, was seized quite suddenly with dimness of vision four days after the commencement of an attack of colic. The amblyopia increased so rapidly that on the seventh day from its first appearance he had but the dimmest perception of mere light.

'O. S. There was a dull grey colouring of both disks, with a decided loss of transparency.

'The powers of vision returned completely under appropriate treatment.'

Hirschler, 'Wien. Med. Wochenschr.' 1866, Nos. 7 and 8, quoted in several journals.

119. *Lead poisoning, optic neuritis.*

(Under the care of Mr. HUTCHINSON.)

Mary W., æt. 19, admitted at Moorfields July 13, 1867. Blind. Pupils large and fixed. Worker in lead mill two years. Four months ago colic, and slightly dropped wrists. Recovery. Fourteen weeks ago vomiting, pain in head, dim vision for five weeks, when she became suddenly blind. She had some numbness in the tips of the fingers.

O. S. 'In both disks were the usual conditions of optic neuritis, being covered with lymph. There were also numerous apoplexies near them.'

Condensed from 'Royal Lond. Oph. Hosp. Reports,' vol. vi. p. 55.

120. *Lead poisoning, dim vision, atrophy of disks.*

I. T., æt. 66, painter, admitted under the author, May 22, 1868. Always healthy, except bad attack of painter's colic, nineteen years ago. Eight weeks ago seized with confusion of head on right side, 'as if a brush had been drawn down that side and left him dizzy.' Had a second attack in a few hours, when he fell. He scarcely lost consciousness but *lost speech*. He quite understood all questions, but was unable to reply. He was also palsied on the *left side*, of motion only. In fourteen days he began to be able to put sentences together. On admission arm chiefly palsied, also left face and tongue. Speech unimpaired. So also hearing and smell and common sensation. Blue line well marked. Improved under appropriate treatment.

O. S. Movements of eyeball normal. Pupils normal or a little

small. Vision for reading dim and useless. Cannot read small print (small pica). Reads large print. No examination of field of vision noted, nor were test types used.

Both retinas normal, vessels rather fine, especially arteries; disks atrophied to the second degree.

121. *Mercurial poisoning, optic neuritis.*

W. W., strong, stout man. Calomel manufacturer. Admitted into Moorfields April 2, 1867. Dim sight five weeks; blindness three weeks. Had been ill four months with pains in limbs, tremors, and much headache. No loss of flesh or appetite; weak and staggering gait.

O. S. Pupils dilated, motionless; media clear; optic disk prominent; vessels protruded and interrupted; edge of disk undefined.

April 8. Delirium; headache.

May 1. Atrophy of disks; still blind.

There is no more evidence given of the mercurialism. T. C. A.

Case condensed from 'Royal Lond. Oph. Hosp. Reports,' vol. vi. p. 54.

122. *Tobacco amaurosis.*

May, 1868.

These two cases the author owes to the kindness of his friend Mr. Oglesby, of Leeds.

Frank G., æt. 50, shoe manufacturer, complains of failing sight. His previous history is good. He has enjoyed good health. He has never suffered from syphilis or other constitutional disease. For some time past he has been mentally depressed, owing to the death of his wife. He suffers from palsy of the hands. He is a great smoker, the average quantity of tobacco he consumes per week being twelve ounces. Rather more than two years ago his vision became imperfect, and has continued to fail somewhat rapidly up to the present time. The ophthalmoscope disclosed commencing atrophy of disks, which rapidly increased, and ultimately resulted in blindness.

Also

123.

Dec. 1869.

J. M., æt. 40, circus-manager, complains of inability to follow his business on account of failing sight. He is irritable and nervous.

Has hitherto enjoyed excellent health. He has been in the habit of smoking the enormous number of twenty-seven cigars per day for a length of time, but was not aware that any injury to health was likely to follow such a habit. Vision is extremely imperfect. The disks are brilliantly white. There is slight palsy of the hands, and great nervousness. I have been unable to watch the case further.

THE tables which conclude the Appendix are the reports of a long and arduous series of observations made upon the insane, chiefly among those in the West Riding Asylum, under the care of Dr. Crichton Browne, and in the North and East Riding Asylum, then under the care of Dr. Christie. The diagnosis in each case is given by Dr. Browne or by Dr. Christie. These tables were first published in the fifty-first volume of the 'Medico-Chirurgical Transactions,' in the hope that the indications of the ophthalmoscope may offer some test by which the alienist physician may sift the unwieldy mass of diseases with which he has to deal, and may approach a more certain knowledge of the various pathological conditions of his patients. Individually the cases are only valuable when verified by autopsies, but taken collectively they seem worthy of republication.

INSANITY WITH EPILEPSY.

Of *insanity depending upon epilepsy* I have noted forty-three cases. It may surprise some of my readers to be told that when compared with general paralysis, mania, and dementia, the proportion of epileptic cases which present symptomatic changes in the eye is small.

I note disease of the optic nerve or retina in fifteen cases out of the forty-three. I mark nine as doubtful ; the remaining nineteen showed no diseased change.

I have said, however (vide chap. v. p. 82), that epilepsy alone (not dependent upon organic disease) is not usually accompanied by disease of the optic nerve, but that a change in the vessels of the retina may be seen at times in epilepsy, and some of the following cases seem to bear this out. On an examination of the table it will be seen that organic disease was known by unilateral symptoms, or otherwise, to exist in most of the cases in which the optic nerves are noted as diseased.

INSANITY WITH EPILEPSY.

Cases of especial interest—13 and 41, 22, 24, 27, 30, 33, 36, 37.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
1	M. Darwin, æt. 25, Wakefield Asylum	Old epilepsy and dementia	Disk a little too pink; edges a little indistinct. No marked change	Venæ centrales very large in this eye; otherwise as right.
2	B. Feathers, æt. 39, Wakefield Asylum	Epilepsy five months; dementia	A little infiltration around the margin of disk	Edges of disk very irregular, and a good deal of infiltration about them, especially toward apparent outer edge.
3	Wm. Hy. Ellis, æt. 3, Wakefield Asylum	Epileptic; enfeebled faculties	Disks, or at any rate right disk, unmanageable.	As right, but less in degree.
4	Chas. W. Walton, æt. 33, Wakefield Asylum	Epilepsy; blow on the head and exfoliation; loss of portion of cerebral substance	Inner edge of disk invisible, of suffused and vascular appearance; other edge clean cut and whitening; <i>vide</i> Lieb. 'At.' Pl. xi. fig. 14.	As right, but worse.
5	M. Evers, æt. 32, Wakefield Asylum	Confirmed epilepsy; much ocular ataxy	Disk pink, about the colour of the choroid, and difficult to find except by convergence of vessels; edges very indistinct; vessels full, not tortuous	

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
6	Wm. Woodhead, æt. 41, Wakefield Asylum	Confirmed epilepsy with de- mentia; tubercle in both lungs	Atrophied; edges clean cut	Some infiltration and excess of vessels on inner edge; other edges, white atrophy sharply de- fined.
7	Elizabeth Crackle, Wakefield Asylum	Confirmed epilepsy and de- mentia Examined again after three or four days of excitement	First examination, healthy Second examination, healthy	First examination, healthy. Second examination, healthy.
8	Martha Dickenson, Wakefield Asylum	Epilepsy occasional; partial paralysis of right side; no aphasia	Exophthalmos; disk healthy? Vessels a little large	Exophthalmos preceded that of right eye, but now receded. Vessels finer than usual. Disk a little whitish?
9	Hannah Gates, æt. 23, Wakefield Asylum	Is liable to long series of fits, followed by long coma. No unilateral spasm	Healthy	Healthy.
10	Sarah Walsh, æt. 55, Wakefield Asylum	Confirmed epilepsy and de- mentia. Late, impairment of muscular power	Healthy	Healthy. A small pigment patch, probably congenital, upon retina.
11	Sarah Holdsworth, æt. 20, Wakefield Asylum	Severe (traumatic) epilepsy; profound dementia. Depression 1 x 2 inches at longest diameters at anterior and upper border of right parietal bone	Disk too pink; edges invisible	Disk natural, unless, perhaps, some slight excess of pinkness.

12	Louisa Sellers, æt. 23, Wakefield Asylum	Epilepsy five years' standing and dementia	There is a whitening at part of margin in both disks, but probably only commencing myopic change.
13	Charles Wood, æt. 27, Wakefield Asylum	Chronic epilepsy and mania, not meningitis. Second examination, has just passed through a violent epileptic paroxysm; has visual and other hallucinations	First examination healthy; vessels large and full. Second examination. On former examination the vessels were noted as rather full than otherwise, but now are very small, fine, and few First examination, healthy? Second examination, vessels here also smaller than usual, but artery and vein distinctly visible.
14	— Scholefield, Wakefield Asylum	Epilepsy and dementia; epilepsy from childhood; left hemiplegia from childhood	Compare this with Case 41.
15	Mary Cooper, Wakefield Asylum	Epilepsy; dementia; phthisis	Disk of irregular form and almond-shaped Otherwise healthy. As right.
16	Hen. Bence, Wakefield Asylum	Epilepsy; dementia	Vessels a little too distinct, and disk rather too pink. Healthy? Disk pinkish? Disk as right; vessels fuller than in right, and more than usually full.
17	Isaac Kendrew, æt. 20, Wakefield Asylum	Continual epilepsy and dementia	Disk atrophied As right.
18	Thos. Greenwood, æt. 30, Wakefield Asylum	Epilepsy and dementia	Much pigmentation about the margins of both disks, which, however, are probably quite healthy.
19	Chas. Paget, æt. 30, Wakefield Asylum	Severe epilepsy and dementia; anæsthesia and impairment of muscular power	Over vascularity and pinkness of disk, but no decided change As right.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
20	Edwd. Scholefield, æt. 20, Wakefield Asylum	Epilepsy and dementia; hemiplegia of left side with wasting of muscles	Healthy	Healthy
21	Jane Sheldon, Wakefield Asylum	Epilepsy and dementia	Healthy	Healthy
22	Rob. Boasman, æt. 33, Clifton Asylum	Dementia, unilateral (right); epilepsy; no aphasia	Commencing atrophy of disk	Perhaps there may be slight atrophy; the disk has a doubtful whitish look, but is not nearly so white as right.
23	Wm. Eggleston, æt. 22, Clifton Asylum ¹	Mania and epilepsy; <i>suspicion</i> of organic disease	Right disk decidedly a little whiter than left	Left is also pale, but I would not assert that either disk is atrophied.
24	Geo. Monkman, æt. 35, Clifton Asylum	Dementia and epilepsy; probably organic disease	Disk congested, and vessels many, but I should not, except for left disk, have attached much weight to it	Left disk is very decidedly congested, and its edges for about half circumference on (apparent) outer side misty. Taking both eyes together there is evidence of change. Cf. Lieb. 'At.' Pl. xi. fig. 14.
25	Joseph Williamson, æt. 22, Clifton Asylum	Epilepsy; dementia; much stupor	Right disk, &c., congested; vessels frequent	Healthy.
26	Rebecca Braithwaite, æt. 36, Clifton Asylum	Mania; epilepsy affecting right side; supposed organic disease	Healthy	Healthy.

¹ No. 23, Wm. E.—, died January 24, 1868, of repeated epileptic seizures. Autopsy forbidden.

27	Hannah Humble, æt. 42, Clifton Asylum	Recurrent mania and epilepsy	Both disks and neighbourhood showing the pinkness and vascularity I have often noted after attacks of mania.
28	Martha Hutchinson, æt. 27, Clifton Asylum	Epilepsy and mania; no unilateral symptoms	Healthy. Healthy.
29	Thos. Storey, æt. 41, Wakefield Asylum	Epilepsy and dementia	Healthy. Healthy.
30	Sam. Jowitt, æt. 31, Wakefield Asylum	Dementia and <i>unilateral</i> epilepsy	Commencing atrophy of disk Atrophy more advanced than right, but scarcely to be called complete.
31	Horatio Edwards, æt. 27, Wakefield Asylum	Dementia and epilepsy	Healthy. Healthy.
32	George Lawton, æt. 28, Wakefield Asylum	Dementia and epilepsy	Disks healthy? Several (congenital?) patches in both eyes about their margins.
33	John P. Halligan, æt. 56, Wakefield Asylum	Eleven years' standing dementia and epilepsy, following depression of skull from a blow (sclerosis?)	Decided atrophy Vision obviously impaired. Complete atrophy.
34	Fr. Richardson, æt. 19, Wakefield Asylum	Dementia and epilepsy	Healthy. Healthy.
35	Alfred Swaine, æt. 26, Wakefield Asylum	Dementia; epilepsy	Some over-pinkness and vascularity in and about both disks, but no marked change.
36	Isaac Challoner, æt. 49, Wakefield Asylum	Dementia or epilepsy, with evidence of cerebral degeneration	Commencing atrophy; outer edge pearly; opposite edge suffused, and vessels many and fine; Lieb. At. Pl. xi. fig. 14. Decidedly whiter than right disk.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
37	Thos. James, æt. 17, Leeds Infirmary	Dementia and epilepsy; uni- lateral (left) symptoms (tumour?)	Has been under my notice for eighteen months, the dementia slowly creeping on. He had, when I first saw him, congestion and exudation in and about both disks. This is now passing into consecutive atrophy with ill-defined edges, &c. Examination, nine months later, shows the edges clearing and becoming definite.	
38	Br. Dyson, æt. 21, Wakefield Asylum	Dementia and epilepsy	Both disks white, vessels few; some indistinctness of inner edge in both.	
39	Alfred Forbisher, æt. 16, Wakefield Asylum	Congenital imbecility and epilepsy.	Disk atrophic at apparent inner edge; many tortuous vessels about opposite edge, but it can be distinguished with $3\frac{1}{2}$ ", not with 2"	Disk atrophic at outer edge; inner edge cannot be distinguished even with 4"; probably some little exudation; one fine and curly vessel crosses the atrophied half
40	Mary Slater, æt. 21, Wakefield Asylum	Dementia, epilepsy; hereditary; father and sister	Vessels to both disks look rather large and a little tortuous, and the disks are not as distinct as they usually are in normal states.	
41	Eliz. Brown, Wakefield Asylum	Dementia and epilepsy. Has just passed out of a status—a succession of fits and coma	Disks anæmic (or slightly atrophic?). She can read No. 1 Jäger. Two vessels of right disk look a little tortuous.	
42	Alfred Burrows, æt. 22, Wakefield Asylum	Dementia and epilepsy	Vessels very fine, almost like embolism	Disk decidedly whiter than right disk. Anæmic?
43	Geo. Ainley, æt. 44, Wakefield Asylum	Dementia and epilepsy	Disks, &c., healthy.	

MANIA.

Of mania I have noted fifty-one cases. Of these, the state of the nerves was, in twenty-five cases, symptomatic of intracranial disease; in thirteen cases it was of doubtful meaning; and in thirteen cases I found either no changes at all or only local changes, such as glaucoma, myopia, &c. I think the reader will be surprised to hear of symptomatic change being found and suspected in so large a number of cases. It was far more than I looked for. But it will be remembered, on the other hand, that the patients submitted to me were always well-marked cases of disease, and in the larger proportion of them organic disease was suspected on grounds independent of my observations. From my list of mania cases I propose the following points for future investigation:

(1) That symptomatic changes in the eye are to be found in a large proportion of cases of mania.

(2) That if cases known to be functional only, or incorrectly named (such as erotomania, transient mania, hysteria, &c.) be omitted, the proportion of cases presenting permanent change in or near the optic disks is still larger.

(3) That both in mania depending upon organic causes and in functional mania the back of the eye, if observed within a few days after a paroxysm, presents a vascular suffusion or pinkness, a pinkness so great after severe paroxysms as to obscure the disk. No exudation is seen in these cases, unless there exist some permanent mischief.

(4) That during the paroxysm, on the contrary, the disk is anæmic, perhaps from spasm of the vessels.

(5) That the permanent changes in the disk are due either to stasis from obstruction to the intracranial circulation, with consecutive atrophy; or to *ramollissement* ending in simple white atrophy; or they may present changes of a mixed character.

I may make a few further remarks upon my propositions concerning the hyperæmia or blush of the disk and fundus in mania. I found this so frequently in maniacs who had recently passed through a paroxysm, that I began to think I could detect cases of mania by this appearance only. However, I carefully

avoided any theorising during my investigations, lest I should unwittingly vitiate my observing powers. On reading my lists over, however, it occurred to me that this suffusion, which I also saw sometimes upon the conjunctiva, might be due to a paralysis of the vaso-motor nerves.

I instantly turned to the notes of a case which I had by good luck been able to observe during a paroxysm, a piece of rare good fortune (*vide* case 31).

I had there noted an anæmic retina, and a whitish state of the disk which I put down to commencing atrophy, though with some doubt. May there be in the eye, and so in the brain, a spasm of the arteries—an epilepsy of the mental functions—followed by a paralytic dilatation of longer or shorter duration? Of course my one case can be nothing more than a suggestion of such a mode of action. The appearances of excessive vascularity seemed to last, on an average, from five to eight days. I watched its fading in several cases.

MANIA.

Cases of especial interest—2, 4, 10, 12, 22, 24, 25, 26, 27, 28, 31, 35, 38, 39. Mania after rheumatism—51.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
1	John Kendall, æt. 44, Wakefield Asylum	Mania passing into dementia <i>Autopsy.</i> —‘Chronic degeneration of brain.’	Simple white atrophy, with even edges This patient is quite blind.	As right.
2	W. Beaumont, æt. 48, Wakefield Asylum	Chronic mania; subacute meningitis? Three months afterwards I hear has had clot on left side, probably in the hemisphere. Previous, therefore, perhaps softening <i>Autopsy.</i> —General wasting and disorganization of brain; atheroma of vessels and clot; weight of brain 42½ oz. This patient died after an apoplectic seizure.	Disk atrophied, edges patchy	Disk atrophied; edges irregular, and patches about and near them, probably of old exudation.
3	Kate Karanagh, æt. 17, Wakefield Asylum	Hysterical mania; severe case This patient completely recovered	Disks both healthy, but perhaps a little too pink; certainly pinker than average	
4	Walter Lee, æt. 14, Wakefield Asylum	Three or four attacks of recurrent mania; meningitis Discharged recovered	Disk suffused; vessels large, plum-coloured, and numerous Certainly some obstruction to the circulation of the retina.	As right.
5	Henry Brown, æt. 45, Wakefield Asylum	Chronic mania	Cornea opaque	Disk, &c., natural.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
6	Jos. Hall (No. 1 ward), æt. 44, Wakefield Asylum	Chronic mania; partial paralysis <i>Autopsy.</i> —‘General wasting of brain; weight 42½ oz.’	Disks rather white at the edges, but probably healthy.	
7	Joseph Hall (No. 2 ward), æt. 75, Wakefield Asylum	Chronic recurrent mania, passing into senile dementia	Disk atrophic; consecutive atrophy, with ill-defined edges	As right.
8	Benjamin Holt, æt. 60, Wakefield Asylum ¹	Chronic mania	Vessels large, and some tortuous; disk too white	Vessels large; disk too white; no infiltration.
9	William Brown, æt. 47, Wakefield Asylum	Chronic mania; constant excitement four months; chronic meningitis <i>Autopsy.</i> —‘Wasted brain; weight 43 oz.; atheroma of vessels; hard, on upper surface of corpora quadrigemina; pia mater glued to layers of arachnoid, but separable from brain. Death occurred during an apoplectic fit.’	Normal? Appearances doubtful	Outline indistinct; some infiltration; vessels well marked, but not much swollen.
10	George Crossley, æt. 34, Wakefield Asylum	Convalescing from a most violent attack of mania. Now has great disturbance of vision, flashes of light, &c., also visual hallucinations	Very deeply congested; vessels many and full; disk only to be recognised by entrance of retinal vessels, where there is a white spot	As right, but even more marked in degree.
11	Thomas Camm, æt. 18, Wakefield Asylum	Mania and morbus cordis	First examination, slightly congested Second examination (six weeks later), right as before	First examination, photophobia. Second examination, still some photophobia, but disk seen, which is more congested than right disk. Almost as right; no important difference.
12	Joshua Kitson, æt. 35, Wakefield Asylum.	Mania; chronic meningitis Died some months subsequently	Atrophied; edges not clean cut, nor yet very irregular, but ill-defined	

Pupils contracted.

¹ Same case as No. 26. Examined at an earlier date.

13	Jane Turnpenny, æt. 21, Wakefield Asylum	<i>Autopsy</i> .—Diagnosis verified; thickening and opacity of arachnoid; adherence of membranes; serous effusion; capillary congestion; tubercle in lungs. Bad case of 'erotomania'	Fundus anæmic; disks healthy.	
14	John Spencer, æt. 23, Wakefield Asylum	Mania functional; subsequent complete recovery	Disk very pink, not to be distinguished in colour from the choroid. Central veins well marked and plum-coloured. Localised pigmentary changes.	As right, perhaps a little more marked.
15	Daniel Milligan, æt. 53, Wakefield Asylum	Mania; subacute meningitis?	There is a slight change in the appearance of both disks, but signifies, probably, commencing staphyloma.	
16	Fred. Simpson, æt. 35, Wakefield Asylum	Acute mania (commencing Graves' disease?) <i>Autopsy</i> .—Brain wasted; weight 43 oz.; pia mater and arachnoid thickened, opaque, and adherent to surface of brain. Symptoms of general paralysis set in subsequent to examination.	Fundus anæmic; disks normal.	
17	William Denton, æt. 38, Wakefield Asylum	Chronic mania	No decided change in either eye; perhaps some fulness of vessels, which are rather plum-coloured.	
18	James Richmond, æt. 34, Wakefield Asylum	Recovered from an attack of acute mania	Disk appears abnormally white, but very transitory view obtained	Could not be seen; patient very difficult to manage.
19	Joseph Broughton, æt. 52, Wakefield Asylum	Chronic mania and meningitis	Fundus anæmic; disks normal.	
20	John Beckwith, æt. 42, Wakefield Asylum	Chronic mania		As right.
21	Harriet Lee, æt. 39 (<i>vide</i> Walter Lee, No. 4, her son), Wakefield Asylum	Recovering from mania. (Has also slight Graves' disease?)	Vessels very tortuous and numerous; disk veiled by infiltration The state known as 'optic neuritis,' and clearly not the retinal stasis sometimes seen in Graves' disease.	

Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
22 Charles Chadwick, æt. 53, Wakefield Asylum	Recurrent mania, slight, right hemiplegia	Decidedly atrophic, edges clean cut	Atrophied; a brilliant white nerve.
23 Martin Devine, æt. 35, Wakefield Asylum	Recurrent mania	Back of the eyes presents marked changes, but they are the changes of myopia.	Complete atrophy; edges distinct, not ragged.
24 Michael Wouldhave, æt. 55, Wakefield Asylum	Recurrent mania, no doubt caused by organic disease; attacks very violent	Commencing atrophy; inner edge vascular and suffused, opposite edge whitening and distinct; no infiltration	Commencing atrophy; edges too clean; vessels numerous; a minute punctiform hæmorrhage near the yellow spot.
25 Robert Hazelgrove, æt. 23, Wakefield Asylum	Recurrent mania, often very severe. Is now recovering from a violent attack; he is also congenitally imbecile	Too many vessels, which are also plum-coloured; some indefiniteness of edges	As right, differing but slightly in degree.
26 Benjamin Holt, æt. 60, Wakefield Asylum ²	Chronic mania; right hemiplegia; attack two months ago, followed by profound coma	Markedly diseased; atrophy at outer edge, and suffusion and vascularity of opposite edge	Both disks very pink, and vessels full.
27 Wm. Williams, æt. 16, Wakefield Asylum	Mania transitoria, functional; attack now passing off	Edges indistinct all round, and disk itself as pink as the choroid. Commencing atrophy?	No decided change; vessels a little dark and full.
28 William Broadhead, æt. 43, Wakefield Asylum	Chronic mania, with degeneration of brain		

² This is the same case as No. 8. Hemiplegia had occurred between the examinations.

29	W. F. Seymour, æt. 45, Wakefield Asylum	Mania recent; just admitted	The disks are very pink, but are both alike, and probably, therefore, healthy.	
30	Ephraim Wood, æt. 37, Wakefield Asylum	Convalescing after acute mania. Gangrene of foot	Disk seems a little white, but cannot be called diseased	Decidedly whiter even than right, but I should scarcely like yet to pronounce it atrophied.
31	B. W. Skelton, æt. 23, Wakefield Asylum	Acute mania (severe). By good fortune we secured an examination during a paroxysm	Fundus anæmic; disk rather white, looking like commencing atrophy	As right, except that at one point of the edge of the disk there seems to be some vascularity.
32	L. Henry, æt. 25, Wakefield Asylum	Mania	Fundus anæmic; disks healthy.	
33	James Grey, æt. 40, Clifton Asylum	Chronic mania. with great depression in intervals of excitement	Disk white and much encroached upon by a patch of pigment; some vessels tortuous; probably some local (perhaps congenital) condition	Healthy.
34	Sam. Jackson, æt. 51, Clifton Asylum	Chronic mania, now passing into dementia	Natural	Irregular in outline and whiter than right disk. No remains of any old exudations.
35	Jos. Blackburn, æt. 29, Clifton Asylum	Mania, probably functional; has been very violent; has been tolerably quiet for three days	Much pinkness of and about disk; vessels full	As right, but even more distinctly marked.
36	Sarah Walls, æt. 64, Clifton Asylum	Chronic mania	Disks, &c., healthy.	
37	Thos. Ward, æt. 44, Clifton Asylum	Chronic mania, passing into dementia	Both disks, &c., healthy.	

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
38	Thos. Brighton, æt. 29, Clifton Asylum	Mania violent, with left hemiplegia	Before I was aware of the left hemiplegia I noted that this right disk was much congested, and the retinal vessels many, full, and plum-coloured (obstruction)	Natural.
39	W. G. Broadbent, æt. 36, Clifton Asylum	Old and bad case of mania. No evidence of organic disease; now quite quiet	Disk red, and vessels rather many and full	Even more congested than right, the edges of the disk being almost indistinguishable; no exudation.
40	Mary Wright, æt. 23, Clifton Asylum	Old history of mania and of states resembling catalepsy, but not very well marked; probably organic disease	Commencing atrophy; some vessels tortuous; outer edge very white	As right, but a little whiter.
41	Mary Bellerby, æt. 46, Clifton Asylum	Chronic mania; seldom excited	Healthy	Healthy.
42	Isaac Hutton, æt. 40, Clifton Asylum	Mania; supposed syphilitic meningitis	Slight atrophy, outer edge being white; edges defined	More marked still, but far, however, from being complete; edges a little ragged.
43	Thos. Kelley, æt. 26, Wakefield Asylum	Chronic mania	Disk pinkish, more than natural; some excess of vessels	Cornea opaque.
44	Wm. Bedford, æt. 67, Wakefield Asylum	Recurrent mania (degeneration of brain?)	Disks, &c., both normal.	

45	Jos. Morris, æt. 37, Wakefield Asylum	Mania; paroxysm recently passed by	No marked change in either eye, but right disk whiter than left.	
46	Edw. Rayner, æt. 41, Wakefield Asylum	Chronic mania; subacute meningitis	Disk whitish at outer edge, and some pinkness about the opposite edge, but taken alone should not be pronounced diseased	Disk very different to right disk, being whiter at outer edge and more suffused about opposite edge. Taking the two disks together, I have no doubt of progressive change.
47	Wm. Woodhead, æt. 41, Wakefield Asylum	Mania; chronic meningitis	Disk ill seen, as the patient was very violent, and kicked and spat while being examined. But the vessels are dark and large, and there seems to be effusion, or, at least, much congestion	Disk ill seen, as was the case with right. But the disk and neighbouring retina look cloudy, and the vessels near the disk are somewhat obscured, though not concealed. No doubt this disk will atrophy.
48	Rob. Feathers, æt. 21, Wakefield Asylum	Acute mania, probably functional; attack passed by some few weeks ago	Disk red or muddy, and with outer edge whitish	As right, but less in degree; not quite right in appearance; doubtful.
49	John Cockcroft, æt. 44, Wakefield Asylum	Chronic mania; degeneration of brain	Disks both quite normal.	
50	Henry Wickham, æt. 25, Wakefield Asylum	Recurrent mania, hereditary	Disk small and reddish	Disk whiter than right, and edges indistinct.
51	Abraham Waddington, æt. 35, native of Conoley. Attended by Dr. McNab, of Crosshills. Wakefield Asylum	Admitted with violent mania, sequence of rheumatic fever; is now recovered (Feb. 18, 1868); has tubercle of lungs	Neither can be pronounced White atrophy distinct, though not very extreme; can read No. 6 Jäger with difficulty	Disk atrophied, but not so decidedly as right disk; reads No. 3 Jäger with difficulty.

DEMENTIA.

The next schedule contains thirty-eight cases of *dementia* not connected with epilepsy. Most of them, however, were due to organic disease in the brain, and were picked out for their severity.

Of these I found disease in the optic nerve or retina in twenty-three cases; I found six in a doubtful condition, and nine were healthy. It will be seen that in simple acute dementia (uncomplicated with organic disease), however profound, no changes in the optic nerve are recorded.

I will only note further, that in dementia, where atrophic changes were seen by the ophthalmoscope, I both made inquiries concerning the sight, and, in some cases, tested it. I confirmed my former belief, that statements concerning visual power, whether made by patients, or by their friends, are of no value (*vide Dementia, e.g.* No. 29); and, moreover, that failure of sight, as tested by types, is wholly an oculist's symptom, and bears little proportion to the amount of atrophic or other disease that may be seen in the nerve. Mapping of the field of vision is a most important physician's symptom, but with lunatics this would have been out of the question.

DEMENTIA.

Cases of especial interest—1, 3, 5, 7, 11, 12, 13, 19, 23, 25, 26, 27, 29, 35, 38.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
1	James Dutton, æt. 48, Wakefield Asylum	Profound dementia; paraplegia; can but just walk <i>Autopsy.</i> —‘Dura mater thickened and puckered; brain wasted; weight 44 oz.; vessels atheromatous, and projecting from cut surface;	Marked atrophy of disk	Cornea opaque.
2	Joseph Sykes, æt. 46, Wakefield Asylum	Dementia	Vessels well-marked; eyes healthy?	
3	Jane Dixon, æt. 65, Wakefield Asylum	Senile dementia; partial paralysis of all limbs	Disks both much changed in outline, and rather too white; frequent pigment spots about margin of both; curious blotchy condition of both retinas, especially of left.	
4	— Bancroft, female, æt. 24, Wakefield Asylum	Acute dementia	Both disks, &c., healthy.	
5	Jemima Batty, æt. 23, Wakefield Asylum	Acute dementia and bronchocele	Active change in both disks; much congestion for about three-fourths of margin in both, which is quite concealed by a veil of exudation; there seems a tendency to whiten at both outer edges; vessels many, the <i>smaller</i> slightly tortuous in many cases; <i>vide</i> Leib. Atl. fig. cit.	
6	Thomas Messenger, æt. 57, Wakefield Asylum	Dementia	Disks both atrophic, not very far advanced; vessels fine.	

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
7	John Kendall, æt. 62, Wakefield Asylum	Dementia following mania	Simple white atrophy, with even edges	As right.
8	Benjamin Kemplay, æt. 62, Wakefield Asylum	Old case of dementia; degeneration of brain	No marked change	Slight, but I think decided commencing atrophy.
9	Henry Oldham, æt. 61, Wakefield Asylum	Dementia; left hemiplegia; old clot	Disk normal? perhaps slightly pink and, it may be, a little indistinct towards outer margin	Disk compressed from above downwards, and much whiter than right disk; could not alone be called decidedly atrophic.
10	Nathan Bentley, æt. 38, Wakefield Asylum	Dementia; tubercle	Both disks misshapen and compressed from above downwards; vessels a little too pronounced; disks are both a little too pink, and their edges somewhat indefinite; both, however, are exactly alike, and probably not diseased.	
11	Patrick Carpenter, æt. 50, Wakefield Asylum	Profound dementia, far gone <i>Autopsy.</i> —Disorganization of brain.	Disks both atrophied, and small; vessels fine; both eyes quite alike. This patient is quite blind.	
12	Richard Buckley, æt. 31, Wakefield Asylum	Dementia; right hemiplegia; clot	Disk too white	Disk decidedly atrophied; edges even.
13	Joseph Hall, æt. 75 (of No. 2 ward), Wakefield Asylum	Senile dementia following chronic recurrent mania	Vessels normal Atrophic disk, with irregular edges	As right.
14	Caroline Brooke, æt. 44, Wakefield Asylum	Dementia; chronic disorganization of brain This patient was examined again some weeks later—observation verified.	Vessels rather too pronounced, and some a little tortuous	Disk decidedly too white; atrophic.

15	Benjamin Lister, æt. 49, Wakefield Asylum	Dementia; softening of brain; paralysis of left side from old clot	Healthy	Cornea opaque.
16	William Pepperdy, æt. 42, Wakefield Asylum	Dementia; clot; right hemiplegia; no aphasia	Healthy	Disk slightly but, I think, decidedly atrophic; decidedly whiter than right disk at any rate.
17	Henry Holloway, æt. 21, Wakefield Asylum	Acute dementia; recovering	No marked change, unless it be some pinkness of the disks and a little photophobia.	
18	David Lambert, æt. 32, Wakefield Asylum	Dementia; masturbation	Disks small and pinkish; vessels a little numerous. I think, however, that this is not a symptomatic change.	
19	Mary Ann Hessey, æt. 47, Wakefield Asylum	Dementia and Graves' disease. (Comp. Harriet Lee, No. 21 of Mania schedule). Since dead <i>Autopsy</i> .—'Adhesion of dura mater to the medullary substance (sclerosis?) of the medullary substance; large serous effusions; old pericarditis.'	Disk shows a tendency to atrophy Her sight is known to have been failing for four or five years. Hardness of the cortical substance; hardness of large serous effusions; old pericarditis.	Left disk decidedly atrophied.
20	John Maguire, æt. 60, Wakefield Asylum	Old dementia and degeneration of brain	Disk looks a little atrophied; a pigment ring round one-third of circumference on outer side.	Disk looks, on the contrary, unnaturally pink.
21	Edward Hurst, æt. 31, Wakefield Asylum	Disease as No. 20, with occasional and various paralyses	Disks both a little troubled, as if with former exudation, but no advancing change.	
22	Charlotte Drake, æt. 62, Wakefield Asylum	Senile dementia; disease of capillaries (?), so that the slightest rap upon the arm causes marked ecchymosis, &c. <i>Autopsy</i> .—'Brain soft, wasted, and watery; much atheroma of vessels.'	The media are dull, and the disks and retinas of a dim muddy look, not exactly anæmic in appearance; no apparent change in the vessels.	

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
23	John Emmott, æt. 19, Wakefield Asylum	Dementia; profound anæmia <i>Autopsy.</i> —‘Brain substance generally pale; layers in cortical matter well defined; veins and sinuses much distended with blood; all the veins about the base remarkably full. Phthisis pulmonalis.’	Disk decidedly atrophic at outer edge; the inner obscured and too vascular	Disk decidedly but not extremely atrophic.
24	Joseph Jubb, æt. 60, Wakefield Asylum	Senile dementia	Pigmentary retinitis both eyes; a large increase of vascular ramifications; disks not decidedly abnormal (whitish?).	
25	Thomas O'Brien, æt. 22, Wakefield Asylum	Dementia following meningitis	Disks pink; considerable increase of vascular ramifications; the circulation of the disks is decidedly affected.	
26	— Wakefield Asylum	Partial dementia; clot; left hemiplegia, and some difficulty in swallowing	Both disks congested, and most of the margin (especially at inner edge) of both invisible.	
27	Jacob Rogers, æt. 60, Wakefield Asylum	Dementia, old case; double hemiplegia, with partial recovery; strabismus; several clots, some rather large	Both disks markedly atrophic.	
28	Mich. Bartlett, æt. —, Wakefield Asylum	Dementia; right hemiplegia; aphasia	Disk perhaps a little too white, and, when taken with left disk, may be called slightly atrophic	Disk decidedly atrophic.
29	John Carrier, æt. 48, Wakefield Asylum	Dementia; right hemiplegia; no aphasia	Disk, inner edge indistinct and suffused pink; opposite edge quite atrophic. Eye blind to ordinary type.	Left disk quite atrophic. Stated by self and others that his sight was in no way failing. Eye reads No. 16 Jäger.

30	Ralph Harper, æt. 50, Wakefield Asylum	Dementia; old disorganization of brain	The vessels in and about both disks are fine and few. I have little doubt that atrophic changes are going on in both eyes, left disk being much whiter than right disk; but as myopic changes are also going on, I cannot give a decided opinion.	
31	George Wheatley, æt. 25, Wakefield Asylum	Acute dementia	Disks natural; eyes anæmic.	
32	William Poppleton, æt. 25, Clifton Asylum	Dementia; no history	Commencing atrophy of disk; inner edge indistinct, and opposite edge white	Decided but not complete atrophy.
33	Edward Farrow, æt. 57, Clifton Asylum	Dementia; paraplegia. (Paralysis below the sympathetic region of cord)	Healthy	Healthy.
34	James Tyman, æt. 42, Clifton Asylum	Dementia; organic disease of encephalon; paralysis of articulation (tongue and larynx); remains of right facial paralysis	Extensive syphilitic choroiditis	Decided but not complete atrophy of the disk; choroid healthy!
35	Joseph Pratt, æt. 51, Clifton Asylum	Dementia; organic disease; degeneration of brain	Disk dirty in colour; edges dimmed in places by exudation, but very partial and slight	Disk, edges obscured by wide areola of exudation; vessels <i>not</i> large or plum-coloured, and are hidden as they approach and pass through the exudation region. This is <i>genuine</i> neuro-retinitis.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
36	Richard Waddington, æt. 33, Wakefield Asylum	Blow on head over left parietal; concussion; interval of health, then gradual change in affections—1, in emotions; 2, in intellect. Wasting and loss of muscular power; certainly has organic disease of brain	Whitish in centre, but cannot be pronounced to be diseased	Left disk compressed and decidedly too white.
37	Ann Gallagher, æt. 16, Wakefield Asylum	Profound acute dementia, functional	No marked changes, unless it be some largeness of the vessels.	
38	Dennis Higgins, æt. 65, Wakefield Asylum	Profound dementia; irregular paralysis <i>Autopsy</i> .—Changing clots on surface of brain; total atrophy exactly up to median line of left half of fornix; wasting of thalami on both sides, which were hollowed out; attenuation of corpora quadrigemina and deep brown discolouration of the nates; optic nerves seemed unaffected to the naked eye, so also commissure and tracts. Under the microscope, however, sections of them showed great proliferation of the connective tissue and atrophy of the nerve fibres.		

MELANCHOLIA AND MONOMANIA

In *melancholia* and *monomania*, which I have tabled together for convenience, the relation of events is changed. As a large proportion of these affections are functional only, so I find, on analysing my list, that of seventeen cases, the optic nerve and retina were healthy in ten, doubtful in four, diseased in three cases. Of the *four* marked as doubtful, the case of John Booth (No. 1 of the table), which is noted as possibly a case of commencing atrophy of disk, has since shown symptoms of locomotor ataxy, as Dr. Browne tells me. This event accounts for my suspicion of commencing atrophy. I find in the three diseased cases that Dr. Browne notes 'atheroma of vessels' in one ; in another 'probable organic disease' ; in the third, 'chronic disorganization of the brain.'

I very frequently noted the presence of anæmia of the retina in melancholia.

MELANCHOLIA AND MONOMANIA.

Cases of especial interest—2, 4, 7, 10, 12, 13.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
1	John Booth, æt. 49, Wakefield Asylum	Melancholia; has since shown symptoms of locomotor ataxy	Contracted pupils; disk rather white	As right. I cannot say that either disk is unhealthy
2	Henry Shirt, æt. 51, Wakefield Asylum	Deep melancholia (moral cause)	Disk <i>reddish</i>	As right.
3	G. Crowther, æt. 45, Wakefield Asylum	Hereditary melancholia (suicidal)	Probably both healthy?	
4	Stephen Sagar, æt. 60, Wakefield Asylum	Monomania; atheroma of vessels	Disks whitish, but probably only anæmic; commencement of pigmentary retinitis.	
5	Thomas Pinder, æt. 37, Wakefield Asylum	Syphilophobia and hypochondriasis; has since recovered	A good many full vessels, and some a little tortuous; disk seems whitening at edges, which are very distinct	Vessels less full and fewer; edge of disk indistinct, as if from very slight infiltration; disk slate-coloured; tendency of part of edge to whiten.
6	Hannah Moorhouse, æt. 37, Wakefield Asylum	Religious monomania, with hallucinations of sight and hearing	Both disks, &c., healthy.	
7	Ann Shaw, æt. 45, Wakefield Asylum	Melancholia; scirrhus of mamma	Both disks, &c., healthy.	
8	Mark Vause, æt. 54, Wakefield Asylum	Melancholia (long history)	Profound anæmia of both optic disks and retinas.	
			Healthy	Healthy.

		Melancholia; anæmia and <i>morbis cordis</i> from rheumatism	Disk pink and a good deal of vascularity; edges indistinct	As right.
9	Eliza Parker, æt. 49, Wakefield Asylum			
10	James Longley, æt. 51, Wakefield Asylum	Confirmed melancholia; mas- turbation; probable organic dis- ease	Pinker than natural (healthy?)	Suffusion of colour and vascu- larity at inner edge; commencing atrophy at opposite edge.
11	Patrick Heys, æt. 40, Wakefield Asylum	Monomania of suspicion, with sense hallucinations, especially visual	Disk flattened from above downwards	Disk whiter than right disk (normal?).
12	Christus Seward, æt. 29, Wakefield Asylum	Melancholia, passing into de- mentia	First examination, disks not natural, but the appearances perhaps those of myopia. Second examination, I think there is decided hyperæmia of the disk and retina, not dependent upon any change in the coats of the eye.	
13	Thos. MacNamara, æt. 28, Wakefield Asylum	Melancholia; old case. Chronic disorganization of brain	Inner edge of disk indistinct and vascular; decided commencing change; opposite edge whitening	Scarcely any change? quite different from right.
14	John Brown, æt. 36, Wakefield Asylum	Profound melancholia; heredi- tary	Eyes not anæmic; disks healthy.	
15	Thos. Abbot, æt. 56, Wakefield Asylum	Melancholia; injury to head	Right disk pinker than left disk. Healthy?	A greyish-white spot on the course of one vessel, and a rather larger and very distinct one near the yellow spot not on the course of a vessel. Both are, I think, old hæmorrhages.
16	Benj. Copeland, æt. 46, Wakefield Asylum	Melancholia; pigment in skin all over body	'Pigmentary retinitis' in both eyes. (Liebreich, At. pl. vi. fig. 1.)	
17	Robt. Moorhouse, æt. 35, Wakefield Asylum	Melancholia	Disks and retinas normal.	

IDIOCY.

The next table is one of *idiocy*.

I examined some idiots because I had formed a suspicion from the few cases which had occurred in my own practice, that atrophy of the optic nerves was not uncommon in idiocy (*vide* chap. v. p. 91). It will be seen that of twelve cases I note very decided atrophy of the disks in five, and advancing disease in one, while two may be called doubtful. Whether this be due to encephalic inflammations in childhood, or to whatever causes it may be, I leave to future observers to say.

IDIOCY.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
1	Alfred Crossley, æt. 21, Wakefield Asylum	Congenital imbecility	No marked change in either eye; perhaps some over pinkness and excess of vessels.	
2	Eugene Slin, æt. 19, Wakefield Asylum	Congenital idiocy	Apparently complete atrophy of both disks, which are clean cut and brilliant; seems to see his way about, but is very stupid.	
3	Charles Blackburn, Wakefield Asylum	Congenital idiocy	Disks decidedly atrophic, having a brilliant whiteness; observation, however, difficult, as the patient has tremulous iris, tremulous eyeball, strabismus, and is deaf and stupid.	
4	George Talbot, æt. 16, Wakefield Asylum	Imbecility after acute hydrocephalus in infancy	Both disks markedly atrophic; outlines irregular, but not uneven or ragged; central vessels of natural size.	
5	Joseph Ogley, æt. 44, Wakefield Asylum	Congenital idiocy; dumb	Normal	Normal.
6	Chas. Thos. Watson, æt. 19, Wakefield Asylum	Congenital idiocy	Right disk pinker than left disk I cannot, however, be sure that there is any progressive change going forward.	Left disk may be in course of atrophy, is decidedly whiter than right disk; also left disk is much larger than right disk.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
7	John Newby, æt. 35, Wakefield Asylum	Congenital imbecility	There may be some atrophic tendency, but neither disk can be called diseased.	
8	Walter John Milner, æt. 26, Wakefield Asylum	Congenital idiocy in highest degree	Commencing atrophy of disk Arteriæ centrales beautifully especially in left.	Very decided, if not complete, atrophy of disk. distinct on the pearly ground,
9	Ann E. Haglewood, Wakefield Asylum	Congenital idiocy	Neither disk healthy, but as there is myopic disease apparent, I cannot decidedly say whether there is any atrophic tendency or not.	
10	Joseph Heald, æt. 30, Wakefield Asylum	Congenital idiot	Opaque cornea	Myopic changes only.
11	Thomas Wood, æt. 19, Wakefield Asylum	Congenital idiot; very slight case	Disks, &c., healthy.	
12	Walter Newton, æt. 20, Wakefield Asylum	Imbecility	Disk rather congested, whitening toward apparent inner edge; opposite edge indistinct and with appearance of old infiltration obscuring the margin; vessels many and full	As right. Neither can be called normal.

GENERAL PARALYSIS.

The next schedule contains fifty-three cases of general paralysis, in five of which I find no change in the optic nerve and retina.

Of the remaining forty-eight, I find atrophy of the optic disks in its various stages in forty-one cases, and seven cases must be marked as doubtful. In all doubtful cases I made two or three examinations at intervals of a few weeks.

I note the following points :

(1) That atrophy of the optic nerves takes place in almost every case of general paralysis, and, I may add, of the olfactory nerves also.

(2) That it does not travel down from the optic centres and along the tracts, but attacks the optic nerves as an independent tract of sclerosis.

(3) It often becomes apparent as a hyperæmia of the nerve with slight exudation, but without much stasis—as a ‘red softening,’ in fact. It then whitens, generally from the outer edge inwards, the nerve becoming white and staring, and its edge sharply defined. (Sometimes it takes a slate colour. *Vide* Liebreich, pl. xi., figs. 6, 12.)

From Case 38 it appears that the smaller vessels become fine and very tortuous before they vanish. If there has been decided exudation, the edges are, for a time, uneven, but the ‘punched-out’ look always establishes itself in the end.

(4) The atrophy of the nerve seems to bear no fixed proportion to the ataxy of the orbital muscles seen in general paralysis. This ataxy is probably dependent upon the same causes as the ataxy of the articulating and other muscles.

(5) The nerve changes are generally proportionate to the well-known contraction and dilatation of the pupils. These contract in the early or hyperæmic stage, and dilate as white atrophy succeeds.

(6) As atrophy of the optic nerves can seldom be surely ascertained in the incipient stages of general paralysis, its diagnostic significance, therefore, is not great. Its value lies rather in its important pathological significance.

Appendix to Cases of General Paralysis.

Dr. Browne has been kind enough to forward to me a report of such autopsies as have taken place upon any of the following cases, viz. upon Nos. 3, 4, 10, 12, 14, 18, 19, 26, 33, 47. In all, the membranes were found thickened and the convolutions wasted and water-logged. In all but two atheromatous disease of the arteries is noted.

In No. 3. The corpora striata, thalami, and corpora quadrigemina were 'flattened, as if wasted.'

In No. 4. 'All the cerebral nerves much wasted; the optic nerves, commissure, and tracts, especially, atrophied.'

In No. 14. 'The optic thalami flattened, as if wasted.'

In No. 18. 'Corpora quadrigemina flattened, wrinkled, and softened; cerebral nerves also softened.'

In No. 26. In addition to the usual changes in the membranes and convolutions, the optic nerves were degenerated. Dr. Browne says, 'the nerves appeared quite plump and round, and I should have reported them as healthy but for their *white glistening appearance*. On examination they were quite without consistence, and were chiefly made up of connective tissue and watery fluid.'

In No. 47. 'Optic nerves, commissure, and tracts, white, flattened, and wasted.'

GENERAL PARALYSIS.

Cases of especial interest—4, 8, 10, 11, 13, 14, 16, 19, 21, 28, 29, 35, 37, 38, 42, 47, 49, 52.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
1	William Brear, æt. 36, Wakefield, W. R. L. Asylum	First stage. Second examination (four months later), in the first stage yet, being slow progress	Rather white. Second examination (four months), as before; disk cannot be called decidedly atrophied	First and second examination, a curious instance of choroidal exudation with ramifying vessels, like Liebreich's 'Atlas,' tab. iv. fig. 5. This patch extends from the region of the spot almost up to the disk.
2	William Walker, æt. 37, Wakefield Asylum	Second stage	Normal? Both disks are much smaller than natural.	Normal? Normal?
3	Thomas Wroe, æt. 46, Wakefield Asylum	Second stage	Too white Veins somewhat large.	Too white.
4	William Sneed, æt. 49, Wakefield Asylum	Beginning of third stage	Commencing white. Atrophy distinct	Complete white atrophy.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
5	Richard Webb, æt. 46, Wakefield Asylum	First examination, second stage Second examination (three months later), end of second stage or beginning of third <i>Post-mortem examination.</i> —Membranes thickened. Arachnoid attached to pia, and many vascular connections. Sulci gaping; grey matter shallow and light coloured, in some places scarcely distinguishable from medullary. Many punctæ sanguineæ on section. Medullary substance very watery and glistening. Ventricles capacious and filled with serum. Weight of whole brain $43\frac{1}{2}$ oz.; of cerebellum, pons, and medulla, 6 oz. Optic nerves unfortunately not noted.	First examination, rather white Second examination, as before	Healthy? Second examination, healthy?
6	Joseph Sykes, æt. 40, Wakefield Asylum	Incipient Second examination (in four months), first stage, slow progress	Normal (Perhaps a little coarsely pink. Second examination, normal	Normal. Pupils very contracted.) Second examination, disk decidedly more staring than right.
7	Henry Cartwright, æt. 41, Wakefield Asylum	Second stage, far advanced	First examination, both disks atrophic and retinal vessels fine. Second examination (six weeks afterwards), previous observation verified.	
8	Duke Waddington, æt. 49, Wakefield Asylum	Second stage	Both disks atrophic, very decidedly; fine vessels; pupils very contracted.	
9	James Greenwood, æt. 45, Wakefield Asylum	Second stage Second examination (in three months), much the same	Both disks atrophic? Second examination, disk atrophic, not far advanced	retinal vessels fine. Second examination, scarcely to be called atrophic, but vessels now rather large and plum coloured, and some of them a little tortuous.

10	Louisa Lockwood, æt. 38, Wakefield Asylum	Beginning of second stage	First examination, inner edge indistinct and pink; opposite edge too white Second examination (four weeks after), appearances the same	First examination, edge clearing all round; decided atrophy at outer edge. Second examination, decided atrophy all round the disk.
11	Ann Taylor, æt. 39, Wakefield Asylum	Second stage	N.B.—For illustration of this and all similar cases <i>vide</i> Liebreich's 'Atlas,' pl. xi. figs. 13, 14. First examination, no change that can be decidedly noted; doubtful Second examination, appearances still doubtful; disk seems pinker and edges not so clear	First examination, disk pale, atrophic? vessels seem too fine. Second examination (in six weeks), I am now sure that there is atrophy of this disk.
12	Sarah Maccaby, æt. 46, Wakefield Asylum	Second, passing into third stage Has had several attacks of 'apoplecticform congestion'	First examination, disk an unnatural colour, being exsanguine and slaty-grey in colour; edges indistinct, and little patches (of old infiltrations?) about the borders; <i>vide</i> Lieb. loc. cit. fig. 13. Second examination (in four weeks), disk whiter	First examination, commencing atrophy. Second examination, I find noted 'decided white atrophy.' (This eye seems to be blind.) (This eye seems to be blind.)
13	James Nowlin, æt. 45, Wakefield Asylum	First examination, beginning of second stage Second examination (six weeks later), beginning of third stage	Autopsy.—Brain very much wasted and atrophied <i>generally</i> ; pia closely adherent to pale and shallow cortical substance; ventricles large; and quantity of serum escaped. First examination, disks do not seem quite natural; pupils, however, much contracted and not sufficiently dilated by the atropine. Second examination, nutrition of nerves decidedly impaired; edges indistinct, and disks of a dirty or slate colour.	

	Name, place of observation, and age.	Name and extent of disease.	Right eye.	Left eye.
14	Jonathan Allen, æt. 41, Wakefield Asylum	First examination, second stage Second examination, beginning of third stage	First examination, both disks suffused pink; edges only to be made out on very careful examination; vessels natural size; pupils very contracted. Second examination (in six weeks), disks both decidedly whitening; edges in some places becoming sharp.	
15	John Hyde, æt. 37, Wakefield Asylum	Second stage	First examination, a great many vessels converging to both disks, especially to this; edges a little indistinct; no decided change however Second examination, gives no more positive results.	As right, but less marked; might pass for a normal appearance.
16	Peter Reynolds, æt. 48, Wakefield Asylum	Second examination, advancing in second stage Second stage Second examination (in two months), is still to be called in second stage, but much worse	Both disks scarcely distinguishable except by convergence of vessels; pink, and edges barely to be made out. Second examination (in two months), much ocular ataxy; very decided suffusion and <i>slight</i> exudation on and about both disks; edges invisible; both disks much affected and vessels many, but right the worse.	
17	John Benson, æt. 35, ¹ Wakefield Asylum	Beginning of second stage	First examination, commencing simple atrophy Second examination (four weeks after), first observation verified. No change Simple atrophy, not extreme, but decided	As right.
18	John Plumer, æt. 43, ² Wakefield Asylum	Second stage		Simple atrophy, as right.

¹ 17. When J. B. had advanced into the third stage, his sight was obviously very much impaired.

² 18. J. P. Vision much impaired before death.

19	Goodworth Fox, æt. 33, Wakefield Asylum	First examination, second stage	Edge pink and invisible towards inner side; atrophic and sharply defined on opposite side	An excess of pinkness and vascularity on inner edge, but not to be pronounced unhealthy.
		Second examination (one month later), end of second stage	Second examination, this disk clearing into simple atrophy	Second examination, as before.
20	James Birkin, æt. 34, Wakefield Asylum	Incipient		
21	Mary Ann Sykes (21 ward), æt. 37, Wakefield Asylum	First examination, second stage	Disks apparently normal, but not to be seen very well, the patient being very unmanageable.	
		Second examination (in two months), third stage	Vessels rather large and dark coloured, and disk pinker than natural Second examination, vessels in both disks diminished, and nutrition of both much impaired; they are slaty-grey, with ill-defined edges (<i>vide</i> Liebreich, tab. xi. fig. 13).	Vessels rather large and dark coloured; disk decidedly whitening.
22	William Hanson, æt. 40, Wakefield Asylum	Incipient general paralysis	Choroid very anæmic; disk natural	Anæmic; disk said by self and three independent observers to be decidedly whiter than right disk, but cannot be called decidedly atrophied.
23	Edward Thornton, æt. 35, Wakefield Asylum	Third stage and death	Complete amaurosis some weeks before death.	
24	John Carrodus, æt. 36, Wakefield Asylum	Third stage and death	Complete amaurosis.	
25	William North, æt. 63, Wakefield Asylum	Third stage and death	Amaurosis nearly complete, but can tell light from darkness.	

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
26	Thomas Binks, æt. 43, Wakefield Asylum	Well on in second stage	Disk as pink as choroid; edges unseen; disk found only by convergence of vessels	Decided commencing atrophy; edges not even, but rather an- gular, not jagged, are sharply defined.
27	James Henry Winn, æt. 28, Wakefield Asylum	Second stage	Disk pink, distinguishable only by entrance of vessels (no infil- tration?); pupils very con- tracted	As right.
28	Thomas Kay, æt. 46, Wakefield Asylum	Second stage far advanced	Marked atrophy, invading from outer edge; indistinctness and pinkness at opposite edge, as Lieb. fig. cit.	Disk quite atrophic, white; edges clean cut; vessels fine.
29	Mark Longden, æt. 34, Wakefield Asylum	First stage far advanced	Disk too white; edges clean cut; vessels beautifully designed upon the disk	Whiter still; extreme atrophy, or at any rate fully developed.
30	Mary Menshull	Incipient general paralysis; progress very slow	No marked change in either disk.	
31	Abraham Whittaker, æt. 49, Wakefield Asylum	First stage	Must be called normal, though it is a little suggestive to me of commencing atrophy	Disk quite atrophied, but is surrounded by irregular patches of (syphilitic?) choroiditis.
32	William Woodhead, æt. 41, Wakefield Asylum	Beginning of second stage	Commencing atrophy; con- gestion towards inner edge; other edge whitening	As right, but more distinct.

33	Harriet Brown, æt. 46, Wakefield Asylum	End of second stage, slow progress	No pronounced atrophy; disk a little staring? a small hæmorrhage in the course of a vessel bending round the yellow spot	Is decidedly whiter than right, but neither disk can be certainly called atrophied.
34	William Robinson, æt. 62, Clifton Asylum	End of first stage	Marked atrophy; nerve slaty grey; vessels fine; edges clean cut, but not very even	As right; simple atrophy.
35	James Butler, æt. 44, Clifton Asylum	First stage	Disk very pink, and edges, especially inner, indistinct. It should not pronounce it decidedly changed except for the condition of left disk	Decidedly whiter than right disk, especially at outer edge, the other being still pink. No doubt right is congested and left whitening.
36	Richard Davis, æt. 32, Clifton Asylum	End of first stage	Opaque (traumatic)	Disk decidedly too white, but not completely atrophied. N.B.—There is some little myopic cupping, but not enough to confuse the diagnosis.
37	Michael O'Connor, æt. 55, Clifton Asylum	Second stage	Disk in congestive stage, very pink, and many converging vessels; edge dim, especially at inner side	Disk beginning to whiten; very different to right disk; edges staring, except some dimness at the inner, which is about the tint of natural nerve.
38	Charles Mercer, æt. 30, Clifton Asylum	First stage, in arrest, and seeming quite well	Decided commencing atrophy, with several small tortuous vessels	A little white crescent or 'arcus' on outer edge, but I should not have noted this save for appearances in right.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
39	William Kirton, æt. 37, ¹ Clifton Asylum	First stage, well marked	Some slight indefiniteness about the edge of the disk	Healthy.
40	— Querchet. Recorded by M. Bouchut, 'Du diagnostic,' &c., p. 333	Stage not stated, probably the second	Atrophy of optic disk	As right, and with central excavation; not glaucomatous, but deeply bowl shaped.
41	William Ledson, æt. 40, Wakefield Asylum	First stage, but rapidly progressed to death <i>Post-mortem examination</i> .—'Pia and arachnoid thickened. Brain surface watery and hollowed out into deep sulci and depressions, one especially in course of middle meningeal artery. Grey matter pale and shallow. Medullary very rosy and speckled with rosy patches. Choroid plexus pale and anæmic. Atheroma of basilar artery and other trunks.'	Commencing atrophy of disk	As right, but still more marked.
42	Walter Griffiths, æt. 44, Wakefield Asylum	Third stage, near termination in death <i>Post-mortem examination</i> , January 12th, 1868.—'Five ounces of serum escaped on removing the brain. Arachnoid slightly thickened and opaque near median fissure. Brain soft and watery. Cortical substance pale and shallow. Convulsions shrunk. Sulci gaping. Numerous and persistent punctæ sanguineæ. Large effusion into ventricles. Corpora quadrigemina wasted and softened, rounded outline lost, testes being quite wasted and almost diffident. Optic nerves, tracts, and commissure all wasted and attenuated, and semi-transparent, of a pale-pinkish, bluish-grey colour. The wasting is more decided as the tracts are traced backwards. Olfactory nerves wasted almost away.'	Disk atrophied; vessels natural size, but varicose; edges not 'punched out,' but obscured by some recent effusion. (Patient blind.)	As right, but there were <i>no vessels visible on the surface</i> ; edges as right.

¹ In a letter I afterwards received from Dr. Christie, telling me that the patients I examined at Clifton (Nos. 34—39) were all living, he adds that he has ascertained impairment of vision in Nos. 34, 36, 37, 38, 39.

43	Samuel Blamire, æt. 35, Wakefield Asylum	Second stage	Patient very unmanageable, and inspection very difficult. Disk thoroughly disorganised, having something the appearance of 'optic neuritis,' but not well seen	Disk dusky and no doubt abnormal, but very hard to see.
44	Charles Redfearn, æt. 39, Wakefield Asylum	Beginning third stage	Disk dusky at inner edge, and many tortuous vessels; the other edge whitening. This disk is in an earlier stage than left disk	Contrast very interesting; outer half of disk quite pearly; the other half is traversed by a few fine vessels.
45	John Rayner, æt. 51, Wakefield Asylum	First stage. At commencement under Dr. Clifford Allbutt, at Leeds Infirmary	Nothing marked in either eye save a little congestion of disks, which may or may not be morbid.	
46	Mark Whittaker, æt. 43, ¹ Wakefield Asylum	Second stage	Disk decidedly redder than left disk. There may be a little atrophic tendency about some parts of edge of disk	Disk seems a little redder than natural.
47	John Sykes, æt. 34, Wakefield Asylum	Beginning of third stage	Optic appearances most interesting. Disk whitening in a blotchy way towards outer edge, and a white line creeping round half circumference; from the white it shades into a dusky reddish brown, and so into a fresher pink at the inner edge, which is quite indistinct; edge blurred all round; vessels rather many	Both disks large. Disk whiter; edge more defined; vessels passing brightly and rather numerous over it. The atrophy is scarcely to be called pearly or staring.

¹ 46. M.W. Now advanced in third stage. Vision much impaired. August 24th, 1888.

	Name, place of observation, and age.	Nature and extent of disease.	Right eye.	Left eye.
48	William Parkinson, æt. 40, Wakefield Asylum	Beginning of second stage	Were it not a case of general paralysis I should have to call the disks healthy, though not quite of healthy look. The disks are alike, but both have a hard pearly look at the outer edges. Two other observers called them white before I had pronounced upon them. Over the white parts are one or two fine vessels, rather too thready and a little curly—these are suspicious.	
49	John Maguire, æt. 60, Wakefield Asylum	Second stage	Optic appearances very interesting, the disks whitening evenly all round, and not from outer edge. Disk dirty red or brown and blurred; dirty white ring all round; depth equal half radius; vessels suspiciously fine	Disk still more of the muddy red appearance, probably less advanced than right disk.
50	Joshua Wilson, æt. 39, Wakefield Asylum	Second stage	Disk dusky, but not distinctly diseased	Disk reddish-brown; edges indistinct; a good many vessels. It is decidedly and characteristically affected.
51	Patience Kershaw, æt. 40, Wakefield Asylum	Beginning third stage	Disks reddish; pigment about edges.	Unaffected?
52	Thomas Brown, æt. 44, Wakefield Asylum	Second stage. Only retains a few words, and curiously unable to receive words; though not deaf, cannot understand what is said to him	Atrophy commencing in a slightly different degree in each eye. In this case the whitening seems of the simple kind, invading from outer edge (and not? preceded by any dusky redness, &c.).	
53	William Henry Smith, æt. 35.	Beginning of second stage	Disks perfectly normal.	

The main inferences from these schedules, which are too long to examine in detail, are that in old or severe organic disease of the brain and its membranes, whether degenerative, hæmorrhagic, meningitic, or due to tumours, obvious changes in the eye are to be seen in a very large proportion.

That in so-called functional diseases, however profound, such changes are to be found only in a very small proportion.

That atrophy of the disk is a common if not a constant symptom of general paralysis, a fact of the highest pathological importance.

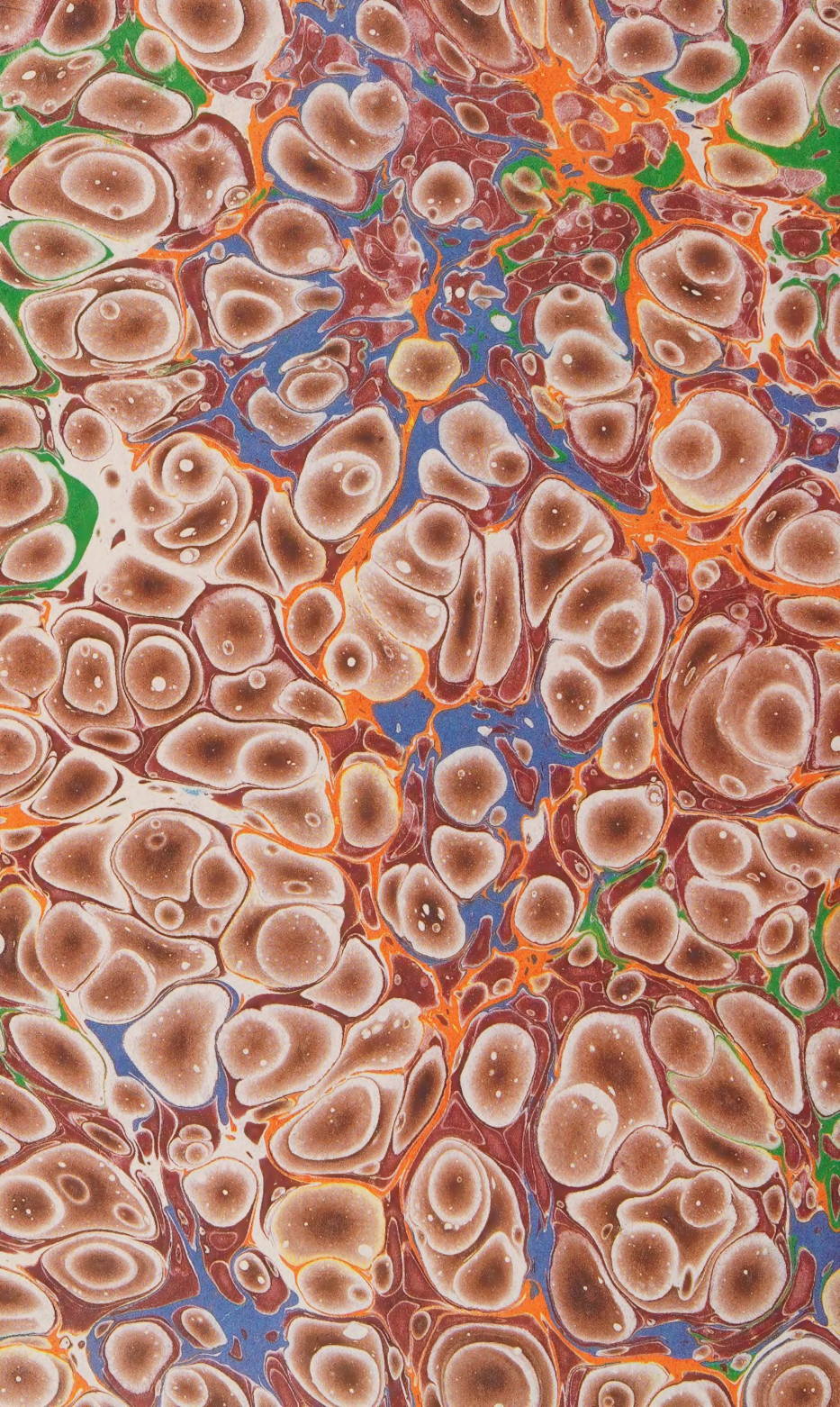
That in mania and epilepsy, but especially in the former, the variations of vascular tension in the retina and disk may have great interest for the observer.

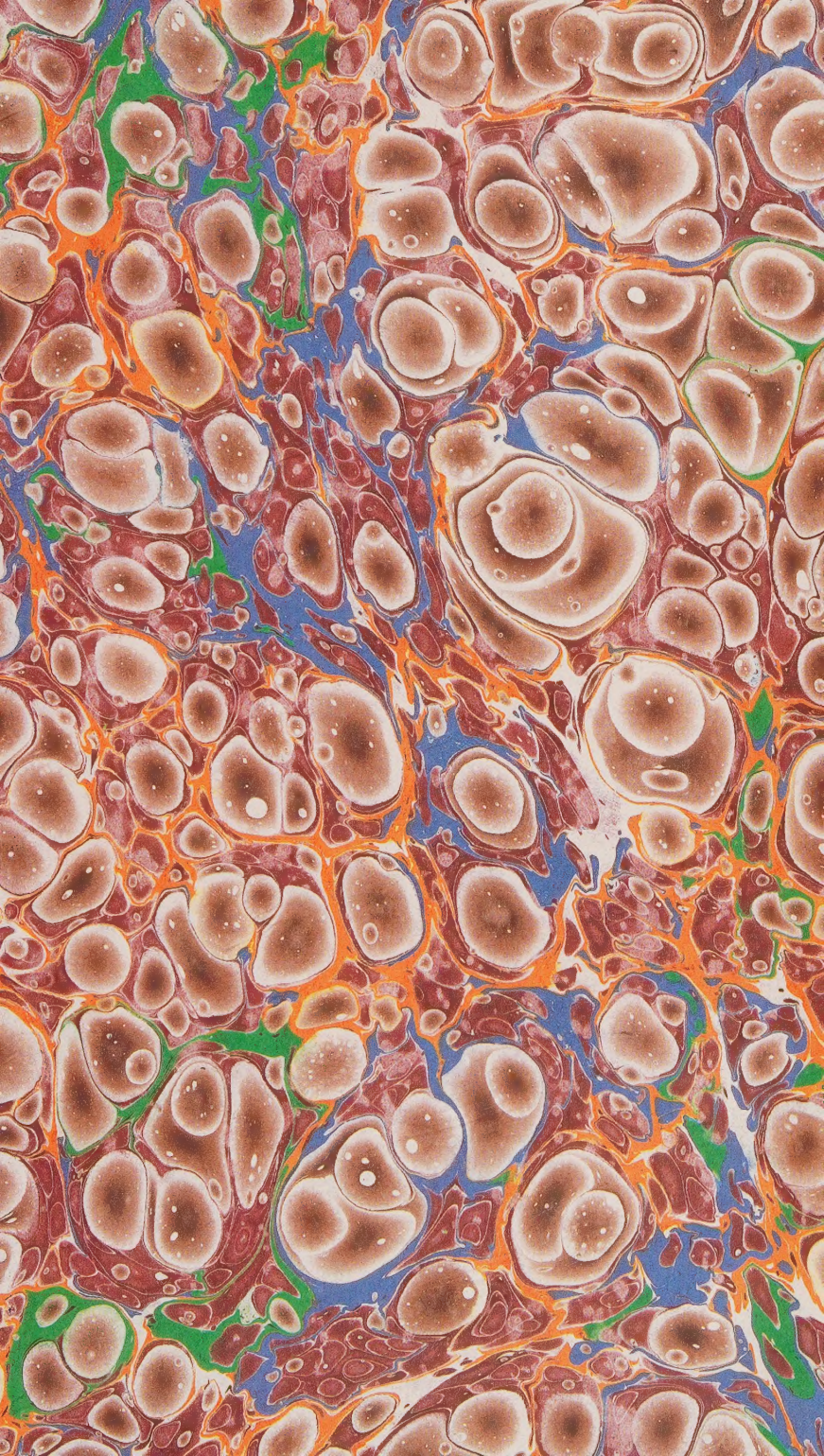


This special edition of

ON THE USE OF THE
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OF THE NERVOUS SYSTEM
AND OF THE KIDNEYS; ALSO IN
CERTAIN OTHER GENERAL DISORDERS
BY THOMAS CLIFFORD ALLBUTT

has been privately printed for the members of The Classics of Ophthalmology Library by The John D. Lucas Printing Company. Film was prepared from the first edition of 1871 courtesy of the Bernard Becker, M.D. Collection in Ophthalmology. New type matter was composed by P&M Typesetting, Inc. in Century Expanded. The text paper was especially made for this edition by the P. H. Glatfelter Company. The volume has been bound in genuine top-grain cowhide by R. R. Donnelley & Sons Company. Endleaves are a specially commissioned original marbled design of Richard J. Wolfe. Edges are gilded and covers are brass-die stamped in 22-karat gold. Cover stampings and design of the edition by Daniel B. Bianchi and Selma Ordewer.







T2-COD-280